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Volume 239 NOVEMBER 18, 1948 Number 21

MEDICAL ASPECTS OF ATOMIC ENERGY*

SHIELDS WARREN, M.D.†

BOSTON

THE Atomic Energy Commission has been charged by you and your fellow citizens with the responsibility of ensuring that the best use will be made of this new source of energy consistent with the national security as well as with maintaining the secret of the atomic bomb and making it available as a weapon. In such a program medicine naturally has a significant role. There is first the problem of maintaining health of the workers in the varied processes incident to the activities of the atomic-energy program. These hazards range from those of radioactive substances in amounts undreamed of a few years ago to the drab and prosaic fall from a stepladder or the almost routine automobile accident. It speaks well for the foresight, the skill and the industry of those charged with medical responsibility in the Manhattan project days that only two people have been killed by radiation hazards. When we remember that many thousands were exposed for several years to potential but adequately controlled hazards this is a remarkable showing; from the fact that among a handful of people exposed more than twenty years ago to the radium watch-dial paint in New Jersey many times this number of radiation deaths occurred, one can see the progress that has been made.

This progress, like the iceberg, showed but little above the stream of contemporary knowledge in the pre-war days. The patient work of the early radiologists, the biophysicists and the experimental biologists gradually and unobtrusively built up a mass of knowledge on which standards of permissible dosage could be safely built. Today there is less hazard in going into plants of the Commission than there is in crossing downtown streets. The knowledge and patient devotion to duty of the health physicists now maintain safety not only for those within the plants but also for those who might be injured by some accident in the environs of the plant.

Secondly, we have the task of developing ourselves and making it possible for others to develop those forays into intellectual darkness by which new continents of knowledge may be mapped out and developed. To give you some idea of the extent to which work in the field of basic medical and biologic research is being carried on, in addition to millions being spent in the Commission's own establishments, over $1,300,000 has been allocated in the last six months for fundamental medical and biologic research in medical schools and hospitals.

Thirdly, no program can be better than the personnel by whom it is conceived and executed. This country is faced today with the scarcity of trained scientists that harks back to the waste of manpower that occurred in the last war. War is always wasteful but no country in World War II was as careless as ours of its trained personnel or as shortsighted regarding the scientific needs of the future. To help repair this loss — a loss that in the world of today is more serious than that of many army divisions or battles — the Atomic Energy Commission has established a fellowship program reaching through all the sciences, but because of our particular interests I shall discuss only those having to do with medicine, biology and biophysics. The National Research Council, at the request of the Commission, has established a fellowship program with Commission funds that is available not only for the doctor of medicine and for the doctor of philosophy in biology or basic medical sciences but also for the college graduate. The postdoctoral fellowships are for two years; the predoctoral are for one year, but may be renewed. It is hoped that a considerable number of predoctorate fellows will continue on to obtain their M.D. or Ph.D. degree by means of aid afforded them through this fellowship program. This program envisages the training of 100 predoctoral fellows and 75 postdoctoral fellows each year.

Fourthly, the Atomic Energy Commission is providing radioactive isotopes for experimental and therapeutic purposes.

*Presented at the annual meeting of the Massachusetts Medical Society, Boston, May 25, 1948.

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To the biologist this has brought unparalleled opportunity for study of metabolic processes and for providing continuous temporal orientation of vital processes that never before could be achieved. To him it has also brought a means of study of chemical relations almost hopelessly snarled.

To the physician it has brought a means of following in detail the action of drugs and distribution of hormones, and has cast new light on problems of metabolism and structure. It has hinted, but only hinted, at new therapeutic methods. As yet there is little hope that we shall find among the radioactive isotopes a control of infection or a cure of cancer. We have found with radioactive phosphorus palliation of certain types of leukemia, palliation of polycythemia vera and some retardation of plasma-cell myeloma. We have found that radioactive isotopic iodine may be taken up by an occasional cancer of the thyroid gland and its metastases and that some of the isotopes in colloidal form may be taken up by the reticuloendothelial system and thus irradiate tumor cells. There are other isotopes that might be useful but for one reason or another — the inherent toxic quality of the element, its long half-life or the specific localization — are unsuitable for use.

The practicing physician needs at present to be concerned with the problem of the atomic bomb. As one of the best educated persons in his community, and the only one technically qualified to evaluate the biologic effects of an atomic explosion, it is his responsibility to orient the members of his community regarding the problems raised by the hazard of such an explosion — problems that are in part psychologic, in part medical and in part organizational.

The air burst of an atomic bomb has many points in common with that of the more usual types of high explosive bombs, but at much higher intensity. An explosion of either type of bomb produces local heat and an air-pressure wave. The explosion of an atomic bomb also causes liberation of radiant energy ranging the whole electromagnetic spectrum from infrared rays to very short gamma rays, as well as producing a flux of neutrons. In an air burst of an atomic bomb the bulk of the fission products are carried to the stratosphere by the rapidly rising superheated air and gases, and are there so widely disseminated as to exert no dangerous effect under ordinary conditions. Hence, there was little or no residual radioactivity to be reckoned with in the Japanese cities.

In an underwater burst, on the other hand, heat and blast are minor factors, but the bulk of the fission products are trapped in the water and spray deluged over the area, a wide zone thus being contaminated with radioactive materials.

It was this type of contamination that made radioactive the ships at Bikini, some of which are too "hot" radiologically to be of any use even up to the present time. In addition, in an underwater burst there may be activity of fairly short half-life as a result of neutron-produced radioactivity. Outside the immediate zone of the explosion the extent of the radioactive contamination will hinge on the speed and direction of the wind prior to the final settling out of the droplets of the radioactive water.

In the United States, as a result of the fairly high level of education, the ready means of disseminating information and what might even be denominated a national tendency to sensationalism the atomic bomb has been endowed with almost supernatural attributes. It is highly probable that in any threat of use of atomic weapons we ourselves should be more vulnerable than anyone else.

It is in this regard that the doctor can be of particular help in enabling those with less information than he to attain the proper perspectives in this new and startling field. Because the doctor is peculiarly fitted to deal with emergency situations, it is natural that much of the planning for atomic defense will fall upon his shoulders. The details of these plans are yet to be worked out because facts are being constantly added to our store of pertinent knowledge. However, sufficient data are available to enable the broad framework of meeting such a disaster to be sketched out.

This is now being done by the Office of Civilian Defense Planning with the aid and co-operation of the Atomic Energy Commission. For the present, the medical profession can best serve by reviewing its knowledge of the effects of ionizing radiation and the therapy of injuries produced by it as well as placing atomic weapons in their proper perspective not as virtually a supernatural force but rather a means of mass destruction cheap and irresistible but nonetheless subject to definite limitations.

195 Pilgrim Road

THE CLINICAL CHARACTERISTICS OF EARLY DIABETES MELLITUS

SAMUEL B. BEASER, M.D.*

BOSTON

CURRENT descriptions of diabetes mellitus vary markedly — as much as 300 per cent — regarding the symptoms of the disease.[1, 2] Likewise, there is little exact information about its early course. The present survey of the population of a large diabetic clinic† was undertaken to clarify these points.

METHODS

The records of 500 consecutive patients were examined, and 182 with adequate descriptions of the onset‡ of the disease were accepted for study.

TABLE 1. *Clinical Features at the Time of Diagnosis.*

PRESENTING COMPLAINT*	TOTAL NUMBER	PERCENTAGE OF GROUP	PERCENTAGE OF TOTAL
Diabetic group:			
Polydipsia	32	49.0	16.8
Polyuria	30	46.0	15.8
Weight loss	30	46.0	15.8
Weakness	25	38.0	13.1
Pruritus†	23	32.0	12.1
Nocturia	9	14.0	7.4
Polyphagia	7	11.0	3.7
Refractive changes	4	6.0	2.1
Headache	2	3.0	1.0
Anorexia	1	1.5	0.5
Drowsiness	1	1.5	0.5
Patients with onsets with one or more of above	65	100	34.0
(Diabetic) complication group:			
Pains or definite neuritis‡	22	62	11.5
Infection	12	33	6.3
Cataract	2	5	1.1
Patients with onsets with one or more of above	36	100	19.0
Casual group:			
Operations	15	16.9	7.9
Cardiac condition¶	13	14.6	6.5
Chronic headache	5	5.1	2.6
Intermittent claudication	2	2.3	1.1
Health examination	2	2.3	1.1
Central retinal thrombosis	1	1.0	0.5
Miscellaneous	61	69.0	32.0
Patients with onsets with one or more of above	89	100	47.0

*Since many patients had more than one presenting complaint, the totals of both types of onset (190) and presenting complaints exceed that of patients.

†Itching of vulvar, scrotal or other skin areas.

‡Symptoms either proved to be due to definite neuritis or carefully investigated to rule out other diseases and subsiding promptly (usually in several weeks) after institution of diabetic care.

§Due to bacterial or mycotic organisms and including skin, genitourinary tract and feet.

¶Including angina pectoris, myocardial infarction and congestive failure or arrhythmia, or both.

They had all been followed on the wards or in other clinics and had been referred to the diabetic clinic because of diabetic symptoms or glycosuria. In most cases a complete medical history had been taken, but an additional diabetic history was elicited. Of these 182 patients, 113 who were found to have had one or more urinalyses prior to diagnosis (usually on morning post-prandial specimens) were investigated in greater detail. Patients with renal glycosuria were excluded from this study.

RESULTS

Classified according to the types of chief complaint, the patients fell into three groups: "diabetic onset," with diabetic symptoms; "complication onset," with diabetic complications; and "casual onset," with glycosuria alone. It can be seen that the incidence of symptoms can vary significantly, depending on the method of analysis (Tables 1 and 2). However, no one complaint occurred in more than 50 per cent of the patients, and fully 23 per cent had no diabetic symptoms (Table 3). The chief complaints comprised only a minority of elicitable diabetic symptoms, individual symptoms being overlooked, as follows: weight loss, 64 cases (70 per cent of the total); polydipsia, 40 (58 per cent); and polyuria, 39 (57 per cent).

TABLE 2. *Total Diabetic Symptoms Present, Including Both Presenting Complaints and Those Elicited by Further Questioning of All Patients, Regardless of the Type of Onset.*

SYMPTOM	TOTAL NUMBER	PERCENTAGE OF TOTAL ONSETS
Weight loss	94	49.5
Polydipsia	72	38.0
Polyuria	69	37.0
Pruritus	49	26.0
Weakness	46	24.0
Nocturia	44	23.0
Polyphagia	17	9.0
Refractive changes	4	2.0
Headache	2	1.0
Anorexia	1	0.5
Vertigo	1	0.5
Drowsiness	1	0.5

Table 4 confirms statistically the validity of classifying the complications separately from the symptoms of diabetes. This is further substantiated by the variation of their incidence among the different types of onset (Table 3).

Decompensation of carbohydrate metabolism can be defined quantitatively.§ Thus, the patients with the diabetic type of onset are in advanced and the others in partial metabolic failure. There is a definite correlation between insulin need and the degree of metabolic decompensation (Fig. 1).

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†In this group 97 per cent of the patients were over forty years of age[1, 3] at the time of diagnosis, as contrasted with 51 and 65 per cent in other clinics.

‡In this study, the term "onset" refers to the date of clinical diagnosis and institution of treatment.

§Partial or advanced decompensation of the diabetic state is defined as simply the possession of any diabetic symptoms or of the progression of such symptoms to the status of chief complaints.

Insulin need is further shown to be greater in youth (Fig. 2), but advanced metabolic decompensation raises the insulin requirement to a maximum level regardless of age (Fig. 2*B*). The lesser insulin requirements of the older patients may be partly explained by the greater proportion of only partially decompensated patients in that group (Fig. 3). However, there is an independent ameliorating effect of age, as illustrated by the paradox that the older patient with symptoms actually needs less insulin than the younger patient without symptoms (Fig. 2).

TABLE 3. *Differential Characteristics of Types of Onset.*

TYPE OF ONSET	TOTAL ONSETS	PERCENTAGE OF TOTAL ONSETS	INCIDENCE OF PREVIOUS GLYCOSURIA	INCIDENCE OF DIABETIC SYMPTOMS		INCIDENCE OF DIABETIC COMPLICATIONS	AVERAGE AGE
			%	%	no./patient	%	yr.
Diabetic	65	34	60	100	3.2	17.0	53
(Diabetic) complication	36	19	36	47	1.3	100.0	59
Casual	89	47	60	73	1.7	14.5	57
Totals	190	100					
Averages			56	77	—	—	56

TABLE 4. *Coincidence of Other Diabetic Symptoms with Selected Single Diabetic Symptoms or Complications.*

SYMPTOM OR COMPLICATION	NO. OF PATIENTS	PATIENTS WITH OTHER DIABETIC SYMPTOMS
		%
Weakness	49	92
Pruritus	44	88
Neuritis	30	66
Infection	15	40

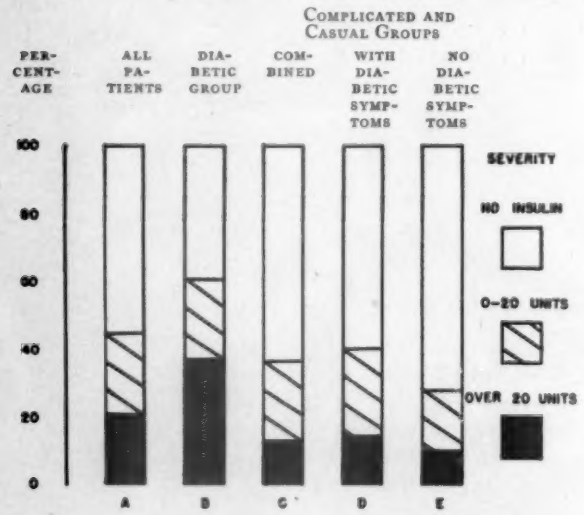

FIGURE 1. *Relation between Degree of Metabolic Decompensation and Severity (Insulin Requirement).*

The insulin requirement was that on a diet of 175 to 200 gm. of carbohydrate and 80 to 100 gm. of protein and fat per day, with minimal glycosuria.

The 113 patients with a record of prior urinalyses were clinically similar to the original group of 182 patients. It is assumed that any previous glycosuria in a diabetic patient with a normal renal threshold suggests the existence of diabetes at the earlier date. Table 5 indicates the progressive increase in incidence of such glycosuria with increasing numbers of haphazard urinalyses performed and the probable diagnostic uselessness of single negative determinations. Accordingly, 13 such cases were eliminated from this study.

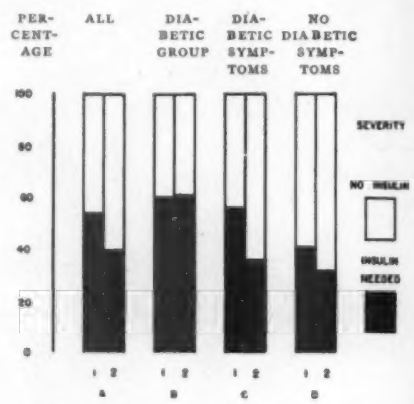

FIGURE 2. *Relation between Age, Metabolic Decompensation and Severity (Insulin Requirement).*

Columns C *and* D *are composed of all patients, including all types of onset, subdivided by symptoms.*

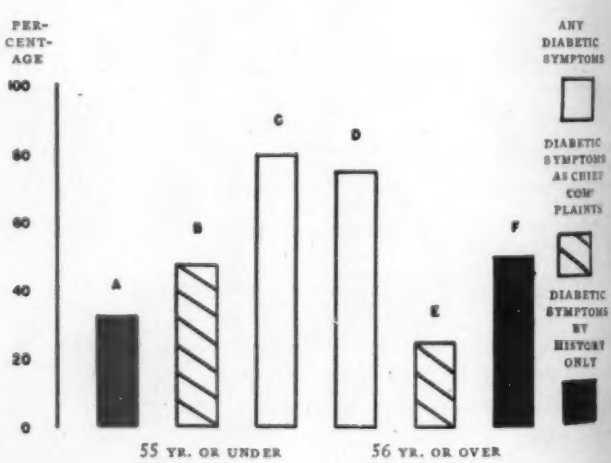

FIGURE 3. *Relation between Age and Metabolic Decompensation.*

Of the remaining 100 patients with previous urinalyses, 56 per cent had had glycosuria. The

other 44 per cent had had none in spite of similar distribution of urinalyses through the years. As an estimate of the duration of the glycosuric stage of the disease, 79 per cent of all the patients with positive determinations had had glycosuria for

TABLE 5. *Relation between the Number of Urinalyses Performed and the Incidence of Glycosuria.*

Total No. of Urinalyses before Onset	Total No. of Patients	Patients with Glycosuria	
		No.	Percentage
1	13	0	0
2–5	48	23	48
6–10	26	13	50
11+	26	20	77

two or more years, and 36 per cent for over six years. Glycosuria preceded symptoms in all cases by two to eleven years (in 33 per cent by over six years).

TABLE 6. *Relation between the Duration of Glycosuria and the Type of Onset.*

Interval before Onset of Earliest Glycosuria	Patients with Diabetic Type of Onset	Patients with Complication and Casual Type of Onset
yr.	%	%
1	32	19
2–5	37	46
6–10	26	27
11+	5	8
Totals	100	100

The diabetic patients had the greatest incidence but the shortest duration of both glycosuria and symptoms (Table 6 and 7).

TABLE 7. *Relation between Duration of Diabetic Symptoms, Glycosuria and Type of Onset.*

Type of Onset	Total No. of Patients	Percentage with Diabetic Symptoms*	Percentage with Glycosuria*
Diabetic	14	100	29
Complication and casual	17	65	18
Total	31		
Averages		73	23

*Duration of one year or less.

The records of the patients with untreated glycosuria showed progressive increase of the glycosuria through the years as well as the acquisition

TABLE 8. *Distribution of Types and Symptoms of Onset.*

Examination of Urine before Onset	Diabetic Onset	Incidence of Any Diabetic Symptoms at Onset	Average No. of Diabetic Symptoms per Patient	Complication Onset	Casual Onset
	%	%		%	%
Glycosuria	32	74	2.0	8	60
Normal urine	30	74	2.2	20	50

of symptoms. Clinically, the patients with negative determinations seem to have arrived at essentially the same end result (Table 8). However, neglect of diabetes in the glycosuric stage resulted in higher insulin requirements (Fig. 4). The incidence of decompensation among patients with both negative and positive determinations was equal (Table 8), so that this factor played no part in the variation of the insulin needs. Actually, prior glycosuria acted synergistically with both advanced and partial metabolic decompensation

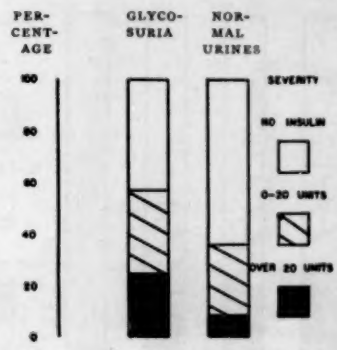

FIGURE 4. *Relation between Prior Glycosuria and Severity (Insulin Requirement).*

in increasing the insulin requirement (Fig. 5 and 6). Yet prior glycosuria seemed to be a more potent factor in this direction than partial decompensation (as shown by comparison of the second and third columns of Figure 6).

To summarize, the factors that are correlated with enhanced insulin requirement, in order of effect, are: a positive history of untreated glycosuria; advanced metabolic decompensation; youth; and partial decompensation.

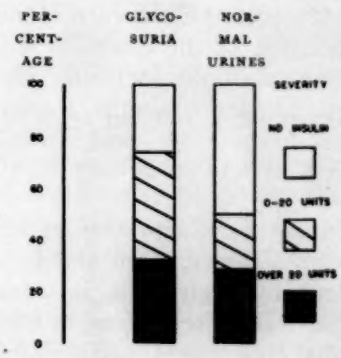

FIGURE 5. *Relation between Prior Glycosuria and Severity (Insulin Requirement) in Advanced Metabolic Decompensation (Diabetic Group).*

The patients with diabetic complications as their presenting complaint were oldest, had less prior glycosuria and required least insulin. Paradoxically, 53 per cent had no diabetic symptoms even though they had complications of the disease (Table 3).

Blood sugar tests had been performed in a few cases in the years prior to diagnosis, and were usually negative because they had been performed in the fasting state. However, 9 fasting blood sugar and 3 sugar tolerance tests had been positive five

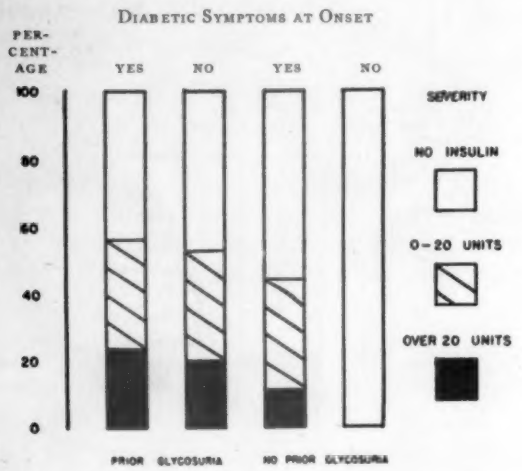

FIGURE 6. *Relation between Prior Glycosuria and Partial Metabolic Decompensation (Complication and Casual Groups Combined) in Their Effect upon Severity (Insulin Requirement).*

to fourteen years before final diagnosis and treatment.

DISCUSSION

It is apparent that more exact classification of the symptoms of diabetes mellitus will help clarify concepts of this disease. Only 34 per cent of cases of advanced metabolic failure corresponded to the textbook description of the disease. A more complete history increased the incidence of symptoms to 77 per cent. However, almost a quarter of the cases remained to be discovered only by means of a glycosuria that was often only mild and intermittent.

True onset must be sharply separated from the clinical onset of diabetes. On the hypothesis of an earlier silent hyperglycemic in addition to a glycosuric stage of the disease (one to eleven years), prediagnosis diabetes would occupy a large portion of the adult life of a majority of diabetic patients. Both these periods may vary markedly in duration. In the purely hyperglycemic period, the individual patients, subjected to the stress of increasing hyperglycemia, react by developing metabolic decompensation or complications, or both. Those with more slowly developing decompensation have time to develop the complications. This explains the paradox of the appearance of a complication of this disease simultaneously with or even before symptoms.[4–6]

The patients with glycosuria showed both increase of their glycosuria through the years and greater eventual insulin requirement than those with negative determinations.

It is conceivable that, if treatment had been started earlier in the course of the glycosuric phase, less insulin would have been required. This may also apply to the prevention of metabolic decompensation, since all 56 of these patients had been seen prior to symptoms.

The patients with negative determinations seem to be heterogeneous. There is one group with a long course, little hyperglycemia and greater tendency to develop complications and a second group with a tendency to rapid development of decompensation once hyperglycemia had appeared (Table 8). In the first, only a glucose tolerance test would have sufficed for diagnosis; in the second, time for diagnosis by any test was probably short. Even though the patients without glycosuria were in the earlier, hyperglycemic phase of diabetes, lack of treatment carried the same poor prognosis (Table 8).

Dolger[7] has raised the somber question whether diabetic complications are inevitable regardless of control, since they eventually occurred in all patients with well controlled diabetes. If the arteriosclerotic complications are included under this heading, this is more than an academic question in an aging population. However, the degree of hyperglycemia in the insulin-treated patient with best controlled disease is probably greater than that in the worst of the untreated patients with complications described above. Yet this group, given time, developed one or more of the diabetic complications. It is highly possible that a patient with diabetes controlled so as to enable him to enjoy normoglycemia at all times will never develop complications, but the disease probably can be so well controlled only in patients detected and treated in the early stage.

Accordingly, from the point of view of preventing metabolic decompensation or of minimizing insulin requirements and diabetic complications, a vigorous attempt to find and arrest diabetes at this early stage is highly important. Although by the age of forty (the youngest of the present group) only 1 out of 6 potential diabetic patients have already been detected,[8] the foregoing analysis indicates that this is none too early to begin the search for the asymptomatic stage of the remainder.

This study shows that in early diabetes single negative urinalyses for sugar do not rule out the disease. More important, even a single slightly positive test obtained under routine conditions, even though apparently overshadowed by one or more previous, simultaneous or succeeding negative tests, should be taken to mean diabetes unless a glucose tolerance test proves otherwise. A fasting blood sugar test is totally inadequate for this purpose.

Summary

Only 34 per cent of a group of adult diabetics had classic diabetic symptoms as presenting complaints at the time of diagnosis. Diabetic symptoms of any type could be elicited from only 77 per cent of all the patients under consideration.

Diabetic complications were shown to be in a different category from the symptoms and were the presenting complaints of only 19 per cent.

There was a lower incidence of severe diabetic symptoms with increasing age.

Severity, as measured by insulin requirement, seemed to be correlated with the following factors: untreated prior glycosuria, metabolic decompensation and age (in order of decreasing importance).

Single, negative urinalyses are of practically no value for the exclusion of diabetes mellitus. Isolated asymptomatic glycosuria, regardless of a negative, fasting blood sugar test, should be considered diabetic unless a glucose tolerance test proves the contrary.

In 56 per cent of a group of adult patients, glycosuria had been noted up to sixteen years (80 per cent over two years) prior to definitive diagnosis. Glycosuria preceded symptoms by two to eleven years.

Detection and treatment in the earliest stage of the disease (hyperglycemic or glycosuric) is urged to prevent progression of severity or complications.

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FATAL TOXIC NEPHROSIS FOLLOWING THE ADMINISTRATION OF MERCURIAL DIURETICS*

Michael S. Bruno, M.D.†

NEW YORK CITY

FATAL toxic nephrosis resulting from the administration of mercurial diuretics is undoubtedly a rare condition. When one considers the extent to which these drugs are used, as well as the failure to find lesions at autopsy that suggest that they have caused ill effects, it becomes evident that morbidity and mortality resulting from their use is infinitesimal.

Friedenson[1] has reported a case in which a total of 1250 cc. of mercupurin (mercuzanthin) was administered over a period of several years to a young rheumatic patient. Autopsy failed to reveal demonstrable abnormalities in the kidneys after careful microscopical study. Gold et al.[2] observed the administration of mercurial diuretics in doses given two or three times weekly for periods of six months to three years and followed 3 patients who had received daily doses for periods of two to three years, without evidence of renal damage. Numerous other reports indicate that mercurials may be used frequently and over long periods without producing renal damage.

The first reported case of fatal toxic nephrosis following mercurial diuretics occurred after the administration of salyrgan.[3] Since then 2 additional cases have been reported,[4, 5] but the diagnosis is not conclusive in either of these cases.

The history of mercurial diuretics since their introduction has been essentially a search for means of reducing their toxicity. Despite all efforts to render the organic mercurials innocuous, however, toxic reactions continue to occur.

Types of Toxic Reactions

Toxic reactions following the administration of mercurial diuretics may be classified in five groups: local reactions; allergic reactions; sudden death; reactions due to the diuretic response; and mercurialism.

Local reactions. Burning, smarting, erythema, local histamine-like reactions and an occasional sterile abscess have occurred after intramuscular administration.

Allergic reactions. Allergic reactions varying from mild to severe episodes have been reported. Fox, Gold and Leon[6] described a case in which the patient reacted severely to the injection of both salyrgan and mercupurin. Apparently, sensitivity to the drug is related to its molecular structure as a whole inasmuch as these reactions can frequently be avoided by employing another preparation. Changing the mode of administration, however, failed to alter both the occurrence and severity of reactions in patients who had previously demon-

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strated hypersensitivity. The occurrence of an allergic reaction should give the physician pause, lest further administration of the offending drug result in more severe reactions and even in death.[7]

Sudden death. This reaction has occurred only after administration by the intravenous route. In 1942 DeGraff and Nadler[8] collected 26 such cases. A number of cases have been reported since that time.[9, 10] In some cases the initial dose was the fatal one, whereas in others, patients had received many previous injections without untoward reaction. Barker, Lindberg and Thomas[11] thought the deaths to be on a cardiac basis, and, after considerable animal experimentation, it was concluded that sudden death was the result of ventricular fibrillation. The opinion was expressed that mercurial diuretics acted primarily on the heart through the mechanism of vagal stimulation, and on the respiratory center if the vagal effect was removed.

Volini, Levitt and Martin[10] reported 3 cases of sudden death following an intravenous injection of a mercurial diuretic, and in 2 of these the electrocardiogram taken just prior to death demonstrated the presence of ventricular fibrillation.

Reactions due to the diuretic response. The diuretic property of mercurial compounds, first described by Saxl and Heilig[12] in 1920, has proved to be a most valuable tool in the treatment of cardiac failure. Mercurial diuretics are the most potent and effective diuretic drugs available to the physician. The urine excreted in response to the drug has a chloride ion concentration much greater than that usually present. Concomitantly, there is a decrease in the serum chloride concentration. This decline in serum chlorides is related to the fact that the kidney under the influence of a mercurial diuretic fails to reabsorb chloride ions in normal amounts, and thus the concentration in the urine is greater than that present in an equivalent volume of body fluid.[13] Occasionally, when there is a profuse diuresis, symptoms related to electrolyte depletion may appear. These symptoms of dehydration may occur despite the presence of peripheral edema. This is probably related to the removal of a large amount of edema fluid containing disproportionately greater concentrations of chloride ions, leaving behind extracellular fluid containing solutes in lower than normal concentration.

Another reaction that has been described is that of digitalis toxicity due to its mobilization from extracellular fluid by the mercurial diuretic. This reaction is probably not nearly so common as is thought, and concern over its occurrence may be obviated by the initiation of mercurial diuretic therapy with smaller doses in fully digitalized patients.

Mercurialism. Poisoning due to the mercury ion per se after the parenteral use of organic mercurial diuretics is rare. Mercurialism is seen most frequently after the administration of inorganic mercury compounds, such as bichloride of mercury. The signs and symptoms of such toxicity are referable to the gastrointestinal tract and the kidney. In the kidney the effect of mercury is to produce a toxic nephrosis in which the damage is localized in the tubules. Although, as mentioned above, organic mercurial diuretics rarely cause mercurialism, probably because the mercury is in a nonionizable organic combination, when it does occur, the signs and symptoms are in no way different from those following the administration of inorganic mercury compounds. Kidney damage after the administration of organic mercurials is exceedingly uncommon, and damage sufficient to cause anuria, uremia and death is rare. For this reason the following case is reported.

Case Report

H. N., a 49-year-old woman, was admitted to Bellevue Hospital for the first time on September 15, 1947, with the chief complaints of generalized edema and severe dyspnea of 1 month's duration.

About 15 years previously, the patient was told that she had diabetes mellitus. The diabetes was well controlled for about 13 years by diet alone, but a symptomatic relapse 2 years before admission necessitated a daily injection of 35 units of protamine zinc insulin. The patient's diabetes remained well controlled thereafter. In December, 1946, the patient had begun to have exertional dyspnea, orthopnea, paroxysmal nocturnal dyspnea and ankle edema. The severity of these symptoms progressed rapidly, and she was referred to the cardiac clinic of another hospital. She was followed at weekly intervals for 3 months and was digitalized and maintained on 0.1 mg. of digitoxin daily. In addition, she was placed on a low-salt and limited-fluid intake and given weekly injections of mercupurin intramuscularly. Despite all therapy the symptoms progressed and in April she was admitted to the other hospital.

Physical examination showed a blood pressure of 210/84 and evidence of hypertensive arteriosclerotic changes in both retinas and distention of the neck veins. The thyroid gland was enlarged to three times its normal size, and there were several hard nodules in the left lobe. A few crackling rales were heard at both bases, and there was evidence of free fluid in the lower half of the right side of the chest. A harsh systolic murmur was heard over the entire precordium and was loudest at the base and extended into the neck. There was also a high-pitched diminuendo diastolic murmur at the base, which was transmitted downward along the sternum to the apex. The liver edge was palpable 2 cm. below the right costal margin. There was no ascites, but there was marked pitting edema of both legs and thighs and moderate sacral edema.

Examination of the urine revealed a +++ test for albumin. The fasting blood sugar was 165 mg., the total protein 6.0 gm. (with an albumin-globulin ratio of 3.4:2.6), the cholesterol 278 mg., the calcium 9.4 mg., and the phosphorus 4.4 mg. per 100 cc. Two phenolsulphonephthalein tests disclosed 70 and 65 per cent excretion of the dye in 30 minutes. Two urea clearance tests were reported as 35.0 and 35.7 per cent of average normal.

The patient was put on complete bed rest and a low-salt and limited-fluid diet. She was continued on 0.1 mg. of digitoxin daily without improvement. On the 14th hospital day, the patient was started on 2 cc. of mercupurin intramuscularly and 6 gm. of ammonium chloride daily. This regimen was discontinued after 3 days because the diuretic response was poor. The patient developed signs of increasing fluid at both bases, and the daily dose of digitoxin was raised to 0.2 mg. In the next few days she began to lose weight. Thoracenteses on three separate occasions yielded 500, 550 and 400 cc. of clear fluid. A daily dose of 6 gm. of ammonium chloride and 2 cc. of mercuhydrin intramuscularly every other day was reinstituted, with marked improvement. Between the 37th and the 39th hospital day, the blood urea

nitrogen rose to 60 mg. per 100 cc., whereas it had previously ranged around 25 mg., and mercuhydrin was discontinued. The patient was discharged to the cardiac clinic asymptomatic, but at the time it was believed that she was entering a terminal phase of uremia.

The patient was seen at the clinic at weekly intervals and was given 2 cc. of mercuhydrin intramuscularly at each visit. Digitoxin, 0.1 mg. daily, was continued in addition to a low-salt diet. Despite all therapy and a marked curtailment of activity, the original cardiac complaints returned within 2 months, and she was readmitted to the hospital. While there she had numerous thoracenteses; 2 cc. of mercuhydrin was administered intramuscularly three times a week, and 0.2 mg. of digitoxin was given each day. She improved somewhat and was discharged to the clinic, where she was followed on the same regimen. In the next 4 weeks the patient developed severe dyspnea, orthopnea, paroxysmal nocturnal dyspnea and anasarca, and was referred for admission to Bellevue Hospital.

Physical examination revealed a poorly developed, well nourished woman in acute respiratory distress and with anasarca. The pertinent physical findings were evidence of hypertensive arteriosclerotic changes in both retinas, a markedly enlarged left lobe of the thyroid gland and distention of the neck veins. There were bubbling rales and rhonchi in both lungs and signs of fluid at the right base. The heart sounds were obscured by the respirations. The cardiac rhythm was regular. The abdomen was tense and distended, and a fluid wave was present. The liver and spleen were moderately enlarged. There was very marked edema of the lower extremities, with moderate edema of the remainder of the body.

The temperature was 98.6°F., the pulse 88 and the respirations 32. The blood pressure was 170/70.

Urinalysis revealed a +++ test for albumin, a trace of sugar, no acetone and a few hyaline and granular casts in the sediment. Examination of the blood disclosed a red-cell count of 4,090,000, with a hemoglobin of 13.4 gm., and a white-cell count of 9400, with 79 per cent neutrophils, 14 per cent lymphocytes, 6 per cent monocytes and 1 per cent basophils. The Mazzini serologic test for syphilis was negative.

It was evident that the patient's condition was grave. She was put on absolute bed rest and a low-salt diet but was given as much fluid as she desired. A daily maintenance dose of 0.2 mg. of digitoxin was ordered. The patient was weighed and given 2 cc. of mercuhydrin intramuscularly and 6 gm. of ammonium chloride. On the 2nd hospital day she weighed 163 pounds. A paracentesis was performed, and 3000 cc. of fluid was removed. This reduced the patient's weight to 155 pounds. Since she had had a good diuretic response the day before, another 2 cc. of mercuhydrin was administered intramuscularly. The nonprotein nitrogen was 50 mg., the chloride 401 mg., the cholesterol 172 mg., and the cholesterol esters 46 mg. per 100 cc. and the carbon dioxide combining power was 29 vol. per cent. On the 4th hospital day the patient weighed 152 pounds but was clinically unimproved. She developed nausea and vomiting, and the digitoxin was discontinued. On the 5th hospital day a slight icterus developed, nausea and vomiting persisted, and a bigeminal pulse was present. The nonprotein nitrogen was 60 mg., the creatinine 1.76 mg., and the total protein 7.2 gm. per 100 cc.; the albumin-globulin ratio was 3.1:4.1. The serum sodium was 306 mg. per 100 cc., and the icteric index was 50.

X-ray films revealed engorgement of both lungs with thickened pleura and evidence of free fluid in the lower half of the right pleural space. On the 6th hospital day a third 2-cc. dose of mercuhydrin was given intramuscularly. The patient continued to excrete urine in large amounts and lost 4 pounds, the weight dropping to 153 pounds. However, the weakness progressed to the point where she could not get out of bed for her daily weighing, and it was decided that her urine output would be checked instead. On the 8th hospital day a thoracentesis was performed, and 740 cc. of fluid was removed.

It was then decided that a more vigorous dehydration regimen was necessary as the clinical course was progressing rapidly downhill. Since mercuhydrin had produced excellent diuretic responses up to this point, it was decided to administer 2 cc. of mercuhydrin intramuscularly every 12 hours. Therapy was continued for 5 days. During this period the patient voided adequate amounts of urine, but she became incontinent and exact measurement of the output soon became impossible. On the 12th hospital day, mercuhydrin was discontinued since the nonprotein nitrogen had risen from 100 to 160 mg. per 100 cc. in a period of 4 days. The carbon dioxide combining power dropped from 36 to 28 vol. per cent. By the 14th hospital day, the patient became incoherent and disoriented. She lapsed into coma and died on the 16th hospital day.

Post-mortem examination 8 hours after death showed moderate icterus and anasarca. The trachea was shifted to the right, and a stony-hard, freely movable mass was palpable in the substance of the left lobe of the thyroid gland. The left pleural cavity was partially obliterated by dense fibrous adhesions; the right pleural cavity contained approximately 1000 cc. of clear, yellow fluid. The abdominal cavity contained 4000 cc. of similar fluid.

The heart was moderately enlarged, weighing 500 gm. There was sclerosis of the leaflets of the mitral and aortic valves and some thickening and shortening of the mitral chordae tendineae. There was some hypertrophy of the left ventricle, moderate sclerosis with narrowing of the coronary arteries and focal myocardial fibrosis. The lungs were congested but were otherwise normal. The liver weighed 1320 gm., and its surface was covered by dense fibrous adhesions.

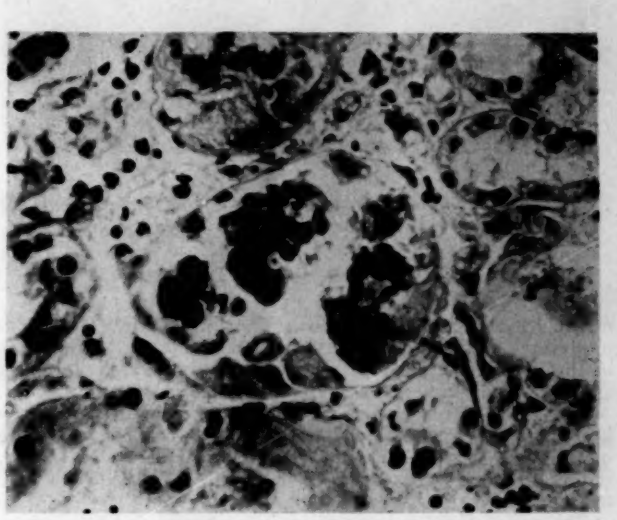

FIGURE 1. *Photomicrograph of the Kidney, Showing Recent Calcium Deposition in the Necrotic Tubular Epithelium (X258).*

About the efferent veins a narrow, red zone was seen. The spleen, pancreas, adrenal glands and biliary tree were normal.

The left kidney weighed 220 gm., and the right 210 gm. There was a deep U-shaped scar in the cortex of the lower pole of the right kidney. Both capsules stripped with ease, revealing a surface that was finely granular. The parenchyma was moderately congested, the cortex was thin, the pyramids were of average size and color, and the corticomedullary junction was well demarcated. The pelvic organs and gastrointestinal tract were not unusual.

The thyroid gland weighed 160 gm. The left lobe was almost completely replaced by a single nodule, which was calcified and well encapsulated. The remainder of the gross anatomic findings were not remarkable.

Histologic examination showed that in the kidney many of the glomeruli had been transformed into compact masses of hyalin. In other glomeruli small rounded masses of hyalin were found at the periphery of the tuft. The basement membrane of the capsule of Bowman was frequently thickened, and the space of Bowman filled with a granular precipitate. Epithelial cells of the proximal convoluted tubules were occasionally seen that were swollen, the cytoplasm being dense and the nucleus pyknotic. Some of these necrotic cells were still in their normal position; others were free in the lumens. Masses of calcium were found in the lumens of these tubules;

in others calcium clumps were present in tubules so altered that it was impossible to identify them. In places the calcium clearly lay in necrotic desquamated epithelial cells and appeared to have been recently deposited (Fig. 1). More frequently the calcium deposits appeared to have been present for a longer time (Fig. 2). Tubules that were lined with flattened cells having a large elongated nucleus and much chromatin were often seen. Occasional mitotic figures were noted in the epithelial cells. The interstitial tissue was moderately increased. Calcium was found to be deposited in the media of a large artery near the tip of a pyramid, and the intima of the vessel was markedly sclerotic.

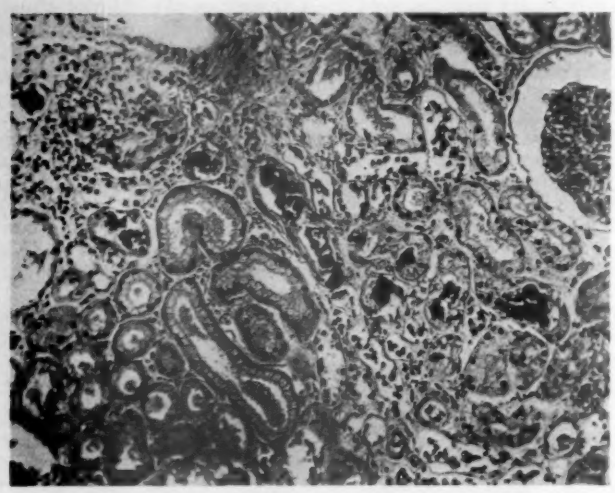

FIGURE 2. *Photomicrograph, Showing Old and Recent Calcification of a Number of Kidney Tubules (X72).*

The Reinsch test for mercury was performed on 10 gm. of kidney tissue, and mercury was found to be present in large quantities. It was estimated that approximately 0.3 to 0.5 mg. of mercury was contained in the 10 gm. of kidney tissue examined.

The anatomic diagnosis of toxic nephrosis due to mercury, with extensive calcification of the kidney tubules, intercapillary glomerulosclerosis and Mönckeberg's sclerosis, was made. Other incidental lesions were those of tuberculous endometritis, biliary cirrhosis, chronic passive congestion of the spleen and lung, focal accumulation of fat in the myocardium, adenoma of the thyroid gland with calcification and hyperplasia of the parathyroid gland.

DISCUSSION

Why fatal toxic nephrosis following the administration of mercurial diuretics should occur remains a puzzling problem. The fact that many patients receive these drugs in large doses for prolonged periods without clinical or autopsy evidence of renal damage indicates that there were unknown factors in the case presented above. Whether preexisting kidney damage contributes to a toxic nephrosis is a much debated point, although much evidence is accumulating that patients with renal impairment respond satisfactorily to the administration of mercurial diuretics.[14] Indeed, as Goldring[15] has indicated, there is no reason for believing that a diseased kidney is more vulnerable than a normal kidney to the nephrotoxic action of mercury: "In fact, it might be supposed that the functionally impaired kidney is less likely to injury, since its inability to concentrate would result in a more dilute solution of mercury in its tubular fluid than in the normal kidney."

What part the large dosage of mercury played in the outcome in this case is also questionable, since such large doses have been given to other patients with renal impairment without ill effect. Although some of the calcification seen in the kidney sections appears to have been of recent origin, much of it seems to have been present for a longer time. This indicates that considerable calcification occurred while the patient was being given smaller doses of the drug as well as when she was on the larger, more recent dosage.

The mercurial diuretics are indispensable in the treatment of many cardiac conditions. The dramatic conversion of a bedridden patient in congestive heart failure to a useful social and economic position in society would often be impossible without these drugs.

In a recent study[16] their importance was emphasized by comparison with digitalis in the control of heart failure, and it was suggested that "in patients with chronic advanced failure who require mercurial diuretics, the mercurial is often the drug of greater importance but that in such cases with auricular fibrillation, digitalis may continue to serve a useful purpose which is not as apparent in those with a normal rhythm." Gold et al.[2] believe "that the sequence employed in the most common practice of trying digitalis first and then adding a diuretic, if necessary, is in need of revision." The basis of their system of therapy rests upon an effective technic for dehydration, with salt restriction, free supply of water and the use of a potent diuretic. The excellent results obtained by this group in the treatment of congestive heart failure, in comparison with routine methods employed by others, testifies to the increasing importance of the mercurial diuretics.

It would be extremely unwise to discard or in any way to limit the therapeutic application of mercurial diuretics because of an occasional toxic reaction. Deaths following the administration of these drugs are extremely rare, and sudden death has occurred only after intravenous administration. There probably exist no contraindications to the use of the drugs other than severe allergic reactions and oliguria or anuria, as in acute glomerulonephritis, lower-nephron nephrosis and other conditions.

Dilution of an individual dose or slowing of the injection time has not reduced the incidence of toxic symptoms.[17, 18] Previous medication with other drugs, such as digitoxin, aminophylline, ammonium chloride and the barbiturates, has not lowered the animal lethal dose.[8]

Probably the surest way to avoid severe toxic reactions would be to avoid administering mercurial diuretics intravenously, except in the few cases of severe congestive failure with acute pulmonary edema in which time is of the greatest importance; to administer a small test dose when the drug is given for the first time; and to change the mercurial diuretic to another drug of this group when an allergic reaction has occurred.

Summary

A case of fatal toxic nephrosis following the administration of multiple intramuscular doses of a mercurial diuretic is reported. Other toxic reactions are discussed.

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PHYSICAL RISKS OF ELECTROCONVULSIVE THERAPY

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ELECTROSHOCK therapy is the most widely used of the somatic methods for the treatment of mental diseases. It is not the purpose of this investigation to describe the therapeutic result achieved or to compare its relative value with other procedures, both somatic and psychotherapeutic. We wish only to evaluate the physical risks involved in this form of treatment. Many papers have been written in the past few years about the subject. Kalinovsky and Hoch,[1] in their excellent textbook, summed up their own experiences and reviewed the literature up to 1945. We hope the close cooperation of our medical and psychiatric staff has made it possible for us to add a few findings of practical value.

This report is based on the observation of 628 patients who received 8082 electroshock treatments at the Boston State Hospital during the six-year period from 1942 through 1947. The only fatal case associated with electroshock therapy occurred early in this period.

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A twenty-three-year-old man, who had been suffering from paranoid schizophrenia for more than four years, died on March 14, 1942, eighteen hours after the eighth treatment in a second series of electroshock therapy. Autopsy by the state pathologist, Dr. Jetter,[2] who reported the case in detail, revealed an acute glomerulonephritis, which was considered to be of several days' duration, acute focal myocarditis and acute pulmonary edema. Physical and laboratory examination prior to the beginning of the treatment was negative.

Because of this unfortunate experience, we became extremely conservative in the selection of patients. Practically every kind of organic disease and physical defect was considered a contraindication to this form of therapy. However, with increasing experience, it became obvious that such an overcautious attitude deprived too many patients in need of the treatment of its benefit. An increasing number of patients with abnormal findings and advanced age were accordingly treated, without ill effect, although this does not mean that electroshock therapy can be used indiscriminately.

A thorough physical examination of every patient preceded the electroconvulsive therapy. It consisted, in addition to a routine clinical exam-

ination, of a 7-foot x-ray film of the chest, urinalysis, complete blood count, a serologic blood test and a determination of the blood sugar and nonprotein nitrogen. The temperature, pulse, respiratory rate and blood pressure were taken prior to each treatment. Additional tests, such as sputum examinations, sedimentation rate and electrocardiograms, were done when thought necessary. Whenever these examinations justified any doubt, the patient was seen by medical consultants to whom we are very grateful for their help and understanding in weighing the risk of the treatment against the expected therapeutic effect. Definite contraindications were found to be rare.

Age

It is now generally accepted that old age in itself is not a contraindication to the use of electroshock therapy. Feldman et al.[3] treated 53 patients over sixty-five years of age, none of whom showed any clinical evidence of injurious effects from the treatment. Thirty-one of our patients were over sixty years old, 4 being sixty-nine and 1 seventy-two. It might be added that during the current year we have already treated 4 patients over seventy. One had diabetes, and several had generalized arteriosclerosis and enlargement of the heart as shown by x-ray study. Marked hypertension was present in 5 elderly patients; the blood pressure was 185 systolic, 110 diastolic, in one, and 172 systolic, 114 diastolic in another. Some had clinical and electrocardiograpic evidence of coronary-artery disease. A sixty-nine-year-old woman had had a coronary thrombosis four years previously while a patient in a private mental hospital. Although she was severely depressed and agitated, electroconvulsive therapy was considered too hazardous at the time. Later, transferred to this hospital, she was mentally unimproved, and electrocardiographic examination confirmed the diagnosis of coronary infarction. Her mental condition was markedly improved after thirteen electroshock treatments. This woman and the other elderly patients withstood the treatment well and developed no complications.

As children have not been admitted to this hospital during the past few years, we can report only 1 case — that of an eleven-year-old girl, who was in an extremely stuporous condition and had had to be fed by tube daily for several months. Six weeks prior to admission, a ventriculogram had been performed in another hospital to exclude organic brain disease. As her mental condition did not improve, she was given seven electroshock treatments, with excellent results. She acted and talked like a normal child and was soon back in school.

Undernutrition

Undernutrition is a frequent finding among mental patients on account of overactivity and refusal to eat. In the preparation of these patients for shock treatment it is often impossible to improve their nutritional condition. However, it is very important not to treat them in a state of dehydration. This can be averted by tube feedings and parenteral administration of fluids if the patient cannot be induced after the intravenous use of sodium amytal to take nourishment. However, too much time should not be taken in preparing the patients, since it is a common and gratifying experience to find them eating ravenously after one or two treatments. The nutritional condition improves so rapidly in these cases that the treatment can be considered a life-saving procedure. One patient, a twenty-five-year-old woman with catatonic excitement, showed extreme emaciation, amenorrhea, loss of hair, hypotension, subnormal temperature and hyperpigmentation so that an endocrinologic disturbance, such as Addison's or Simmonds's disease, was seriously considered. After seven electroconvulsive treatments, she was in full remission; her physical condition improved rapidly and she was able to leave the hospital.

Another patient, a thirty-one-year-old man who was 6 feet tall, and whose weight was down to 83 pounds, in a catatonic stupor, refused to eat and was fed by tube daily for two months. Permission for shock treatment was received from his family only after his weight had decreased so severely that his condition had become critical. After the second electroshock treatment, he began to eat by himself, and his physical condition improved rapidly. Six weeks after a series of fifteen electroconvulsive treatments, his weight was 125 pounds; he too was able to leave the hospital on a visit. In these 2 cases, the shock therapy was life saving. They indicate that severe malnutrition is no contraindication to convulsive treatment.

Secondary anemia, which is often associated with malnutrition, was found in several patients. Even when the red-cell count was less than 3,000,000, the treatment was not excluded. With better food intake after the electroshock therapy, the condition of the blood also improved. When necessary, the patients received iron and liver medication, but electroconvulsive therapy was not delayed by the medical treatment.

Cardiovascular Diseases

Cardiovascular diseases present the most common cause for withholding electroconvulsive treatment from patients in need of it. However, as early as 1942, Myerson[4] pointed out that the damage to the heart from the psychosis itself put a greater burden on the cardiovascular system than the shock treatment. Among our patients

were 27 in whom enlargement of the transverse diameter of the heart was noted on 7-foot x-ray films. Ten of these patients showed clinical and roentgenologic evidence of arteriosclerotic heart disease, often accompanied by generalized arteriosclerosis. One patient, a sixty-two-year-old woman, with a diagnosis of psychosis with cerebral arteriosclerosis, received twelve electroshock treatments with no untoward physical effects. However, she remained agitated and depressed, and lobotomy was later performed. At operation, the neurosurgeon found advanced arteriosclerosis of the cerebral vessels, with severe atrophy of the brain. After lobotomy, her mental condition showed marked improvement. This is a proved case of advanced cerebral arteriosclerosis in a patient who withstood a course of electroshock treatment without physical damage.

Twenty patients had abnormal electrocardiographic findings, varying from simple left-axis deviation, low voltage and an inverted T wave in Lead 4 to signs of definite coronary-artery disease. The case of a woman with coronary thrombosis was described above among the group of elderly patients. Another patient, who was known to have congenital heart disease (patent ductus arteriosus), received nineteen electroshock treatments. Still another patient, a thirty-seven-year-old woman with a severe agitated depression, had rheumatic heart disease with mitral stenosis and aortic insufficiency. She had a history of several episodes of severe decompensation. After five electroshock treatments, she became decompensated again and had to be digitalized. Her mental condition was markedly improved. All the other heart cases showed no untoward physical effects whatsoever.

Marked hypertension was found in 16 patients, 4 of them with a systolic pressure of more than 200. One woman of fifty-eight with an enlargement of the heart on x-ray study had a blood pressure of 220 systolic, 140 diastolic. Her severe, agitated depression, which did not respond to any kind of sedation, made electroshock necessary in spite of the risk. She had seventeen treatments, and like the rest of the patients with hypertension, did not show any ill effect. In many cases the blood pressure dropped to normal levels when, as a result of the treatment, agitation and tension subsided. Seven patients, who showed very little or no mental improvement, had marked hypertension at the end of the treatment although the blood pressure had been normal at the onset. In 1 patient, a forty-five-year-old woman, the blood pressure, which had been 138 systolic, 92 diastolic, before treatment, rose to 174 systolic, 110 diastolic, after nine electroshock treatments. In a few other patients it went up and down in a way that could not be easily understood, since it was, sometimes, inconsistent with the emotional state of the patient. One observation especially appeared to be remarkable. A woman in her middle fifties, in a severe depression very close to complete stupor, had a blood pressure of 158 systolic, 108 diastolic, at the beginning of treatment. After only four treatments she was in full remission, but her blood pressure rose to 220 systolic, 118 diastolic. The patient left the hospital four weeks after the treatment and remained well, both physically and mentally, during a follow-up period of more than a year. Obviously the blood pressure became high when she came out of her stupor; hypertension was normal for her cardiovascular system as soon as compensation was established.

Among 4 patients with hypotension a forty-nine-year-old woman, who had had a blood pressure of 90 systolic, 70 diastolic, showed prolonged cyanosis after the first electroshock treatment. With improvement of her mental condition her blood pressure and pulse pressure went up, and further treatment went on without any incident.

Five or six patients in whom cyanosis and poor quality of pulse persisted for more than a few minutes after treatment had not shown any physical abnormal findings prior to the treatments. They recovered instantaneously after application of oxygen and stimulants.

None of these patients with cardiac findings, hypertension, hypotension or unusual oscillations of blood pressure showed any ill effects from the treatment.

Pulmonary Tuberculosis

The high incidence of pulmonary tuberculosis among patients in mental institutions is well known. The question frequently arises whether or not electroshock therapy should be given. Several cases have been reported in the literature with spread or reactivation of the tuberculous lesions after electroconvulsive therapy. The increased intrathoracic pressure, the pulmonary congestion and the anoxia may all have deleterious effects. However, it should be kept in mind that soft and active lesions are prone to spread anyway. There are no investigations available that would demonstrate whether such activations occur more frequently with or without electroshock therapy. Norman and Shea[5] reported from the Foxborough State Hospital the cases of 2 patients who developed active pulmonary tuberculosis after electroconvulsive therapy. Will and Duval[6] described another case from St. Elizabeth's Hospital in Washington, D. C., and reviewed the whole literature up to 1947.

In our own cases we tried to evaluate the criteria of activity or inactivity as much as possible before giving electroconvulsive therapy. It is obvious that x-ray findings are not the sole basis for the

diagnosis. Whenever the roentgenologic impression indicated a tuberculous process, the patient was seen in consultation by a tuberculosis consultant, and further laboratory examinations such as examination of the sputum or stomach washings for tubercle bacilli and sedimentation rates were performed.

Twenty-five patients had healed pulmonary tuberculosis; the findings in 15 additional cases were considered doubtful. All these patients received routine courses of electroshock therapy without activation of the pulmonary process.

Eight additional patients were considered to have active pulmonary tuberculosis, and 2 probably active. The diagnosis was based on x-ray findings, sedimentation rate and evaluation by the tuberculosis consultant, although acid-fast bacilli could not be found. One of these patients, with a manic-depressive psychosis, had an exudative pleurisy of the left side prior to shock treatment. After a series of fifteen electroconvulsive treatments, her great overactivity subsided, but she developed a pleuritic exudate on the other side. A tuberculous pleurisy often spreads to the other side, and it is difficult to say how much the shock treatment had to do with it. The other 7 patients in whom the examination prior to electroconvulsive therapy revealed a tuberculosis of some activity showed mental improvement or at least less agitation after shock treatment. This may be the reason why their mild tuberculosis is taking a favorable turn.

Another case presented an extraordinarily grave problem: an active pulmonary tuberculosis, of serious prognosis in itself, occurred in a patient who was in a state of severe catatonic excitement. It was obvious that active therapy had to be started immediately. As any therapist in this field may face such a case some day, and will have to make his decision, this case is presented in greater detail:

A 20-year-old Negress was admitted in an active schizophrenic attack, which had started a few days before. Her catatonic excitement became gradually worse; sedation and continuous baths were applied with little success. She refused to eat and had to be fed by tube. She lost weight and had subfebrile temperatures. A chest plate revealed soft areas of infiltration in the right apex and the first right intercostal space. The blood sedimentation rate was increased.

On July 18, 1947, a right pneumothorax was performed by one of us (J.D.W.). The filling was increased 2 days later and resulted in good collapse of the lung. On July 21, she received the first electroconvulsive treatment. Subsequently, every other day she had a shock treatment or a fluoroscopic checking of the lungs with refilling of the pneumothorax, when necessary. Her condition improved steadily. After the first shock treatment she was eating, and the intervals between single treatments could be made longer. Altogether she had nineteen electroshock treatments, the last on September 3. One month later, she was released from the hospital, mentally in full remission and with the right lung in good collapse. At present she is attending one of the tuberculosis units in this city for maintenance of her pneumothorax and is working as an office clerk.

Two patients had had normal chest films prior to electroconvulsive therapy. Both had been in mental hospitals for years. One, a twenty-eight-year-old woman, with chronic catatonia, had five electroshock treatments without improvement. Six months after convulsive therapy, she developed a rapidly progressive pulmonary tuberculosis from which she died three months later. The other patient, a sixty-three-year-old woman with agitated depression and confusion, had signs of cerebral arteriosclerosis. On account of her severe agitation she received eighteen electroconvulsive treatments within a period of ten weeks without improvement. A few weeks after discontinuation of shock therapy, she developed a high temperature; additional chest films showed a bilateral active tuberculosis. The pulmonary infection took a rapid course, and she died four months later. After the development of the tuberculosis, the chest plates that had been taken of both patients prior to shock treatment were scrutinized again and even then the films had to be considered normal. As a certain number of patients with chronic schizophrenia may be expected to develop pulmonary tuberculosis, it remains an open question if and how far the shock treatment should be considered responsible for the activation of the process. In any case, as the chest films prior to electroconvulsive therapy were entirely normal, we had no reason to withhold the treatment in these cases.

In summary our experiences with the problem of electroconvulsive therapy of mental patients who are simultaneously suffering from pulmonary tuberculosis indicate that the treatment carries definite risks. On the other hand, if the patient becomes exhausted by overactivity or refuses to eat, the prognosis of the pulmonary process is in itself very serious. In each case it is necessary to evaluate carefully the degree of the tuberculous lesions, their possible aggravation by the psychosis and the result expected from the shock treatment. There is a good argument for the use of electroconvulsive therapy in selected cases of pulmonary tuberculosis even if some degree of activity is present. If the activity is high or if there is a history of recent hemorrhage, shock should be given only when the pulmonary process has been controlled by some collapse measure, such as pneumothorax. There are cases in which collapse therapy and electroshock have to be applied simultaneously.

Bronchiectasis

Six patients had roentgenologic signs of bronchiectasis without increased secretion. They had no ill effects from electroshock therapy.

Acute Infections

All patients suffering from acute infections should be excluded from electroshock therapy.

Whenever any upper respiratory infection was present, treatment was postponed. The same rule was followed in local infections as abscesses or cellulitis. A few patients with chronic otorrhea were given electroshock therapy without ill effects. These cases were evaluated first by clinical examination, x-ray study of the petrous bones, blood sedimentation rate and white-cell count. When any activity of the ear infection was found, the patients were not treated. If there could be any doubt about the rule that patients with acute infections should not be given electroshock treatment, this was re-emphasized by the only fatal case connected with electroshock that occurred in this hospital. As mentioned above, post-mortem examination in that case revealed an acute glomerulonephritis of several days' duration. One of the 2 fatal cases after electroshock treatment recently reported by Riese,[7] was very similar to that discussed above. A thirty-year-old obese woman died forty-eight hours after electroshock treatment. Post-mortem examination showed subacute cystitis and acute pyelonephritis. If any conclusions can be drawn from cases of this kind, they are that intercurrent infections must be watched for during a course of electroshock treatment as well as prior to it. A urine examination should be made at least once a week during a course of treatment.

Bone Disease, Fractures and Dislocations

Any kind of bone lesion found prior to electroshock treatment is important, since a higher incidence of fractures can be expected in such cases. A few observations confirmed this opinion. One patient, whose x-ray examination had shown narrowing of the interspaces between the dorsal vertebras, sustained a fracture of the third dorsal spinous process from electroshock therapy. Another patient, a thirty-eight-year-old woman, had an ankylosis of the right elbow from an old fracture. During the sixth electroshock treatment, she suffered a fracture of the neck of the right humerus, most probably because too much pressure was exerted on the ankylotic limb during shock.

Observations of this kind should not lead to the exclusion of the needed treatment but should indicate that extreme care is necessary in placing such patients on the table and in exerting the proper amount of force in restraining them. As a matter of fact, in our series, numerous patients with bone disease were given electroshock treatment without damage. (A case of spina bifida is described below in the section on central-nervous-system disorders.) One patient had a severe kyphoscoliosis, and shock treatment had been considered in another hospital as too dangerous. As her catatonic excitement persisted she received a series of electroshock treatments without ill effects. Five patients had scoliosis of moderate degree; 2 had marked arthritic changes of the spine on x-ray study. One woman, thirty years of age, had multiple areas of increased calcification in the frontal region, which was considered roentgenologic evidence of enostosis frontalis. She received fifteen electroshock treatments without untoward result. Six patients had x-ray findings and histories of fractures prior to the treatment: an elderly woman had sustained a fracture of the jaw in an automobile accident only two and a half months before she had electroshock treatment; another patient, a sixty-five-year-old woman, had had a fracture of the right hip one year previously; 3 patients had old rib fractures; and 1 patient had an old occipitoparietal fracture of the skull, with slight bone depression and negative neurologic findings. All these patients had no ill effects from the treatment.

Fractures due to electroshock treatment occurred in 11 patients, including the 2 cases with previous bone disease mentioned above. Of the 9 patients without previous bone disease, 8 sustained fractures of the spine. As other observers have noted, all these fractures healed with amazingly little discomfort to the patient and without orthopedic treatment. One patient, a young man of nineteen in poor nutritional condition, suffered a fracture of the left acetabulum. Unlike the course in the case reported by Myerson,[4] the head of the femur did not penetrate into the pelvis, and there was no marked displacement of the fragments.

One dislocation of a shoulder occurred in a patient who later gave the information that he had had such an injury before. The humerus was replaced immediately without difficulty. In 3 cases dislocation of the jaw was easily reduced.

When the measures necessary to prevent injuries to the skeletal system are considered, the need of proper placing on the table, with one pillow under the small of the back and another under the knees of the patient, should be stressed. Only moderate restraint should be exerted by the assistants who hold the patient. Another measure, often recommended, is the preliminary administration of curare. As the drug has been tried by us in only a few cases, our experience is not sufficient to justify an opinion about this. However, the observations of other workers seem to show that the dangers of this drug outweigh its advantages. During a twelve-month period, ending June 30, 1946, 3 deaths occurred incident to the administration of electroshock treatment in all mental institutions of Massachusetts; 2 of these were associated with preliminary curare medication.[8] Several other fatal cases after the use of curare are reported in the literature. Of the 2 fatal cases recently described by Riese,[7] 1 was mentioned above in connection with acute infections. The other death occurred in a patient who had received curare prior to electroshock therapy. These and similar reports

explain our reluctance to use curare in the preparation for convulsive treatment.

Central-Nervous-System Diseases

A seventy-four-year-old man with seropositive syphilis and a severe depression had a positive cerebrospinal-fluid Wassermann reaction. Another patient, a woman of forty, had residual poliomyelitis. Both had no ill effects from the shock treatments.

Another patient, a thirty-one-year-old woman with a schizoaffective psychosis, who had not shown any abnormal physical findings prior to shock therapy, developed a transient hemiplegia of three days' duration after treatment. The paralysis disappeared without lasting damage to the patient. As there is still insufficient knowledge of the basic changes in the central nervous system caused by electroshock therapy, such a case might be of value toward a better general understanding of the pathology of electrically induced convulsions. In a separate report an attempt was made to explain this transient hemiplegia on the basis of vascular spasm, causing reversible structural damage in the motor pathways of one side of the brain.[9]

Another patient presented a considerable problem in the decision whether to give her electroshock therapy. She was a woman of thirty, who had a sacral spina bifida with a dorsally protruding meningocele the size of a large hen's egg. Extensive involvement of the spinal cord had caused a spastic paraparesis. Her right leg and several toes of her left foot had been amputated. She had dementia praecox with depressive and paranoic features, and was almost continuously disturbed. She was noisy and threw dishes and similar objects at nurses and doctors. Numerous consultations were had for two years, since we hesitated about giving her shock therapy. Finally, when her mental condition became worse and tube feeding had to be given frequently, a course of electroconvulsive treatment was started. Up to this writing the patient has received twenty treatments with no physical ill effects and marked mental improvement. Although some paranoic trends are still present she is well behaved and laughs and talks with everybody who comes to see her.

Menstruation and Pregnancy

Normal menstruation is no contraindication to the treatment. Only when the hemorrhage was profuse did we wait until it diminished. In the literature are several reports of pregnant women who received electroshock treatment without damage to mother or child.[1] We had no pregnant women in our series.

Diabetes Mellitus

Five patients had diabetes mellitus; 1, a woman of sixty with a manic psychosis, had blood sugar values up to 275 mg. per 100 cc. She had an excellent remission after nine shock treatments. She, as well as the other diabetic patients, withstood electroshock therapy without untoward physical effects.

Summary

The physical risks of electroconvulsive therapy are discussed on the basis of a material of 8082 treatments received by 628 patients. Thirty-one of them were over sixty, and 5 over seventy years of age. All these patients responded well to the treatment, as did a child of eleven. Even severe undernutrition is no contraindication to the treatment.

About 50 patients had signs of cardiovascular diseases, such as arteriosclerosis, enlargement of the heart, abnormal electrocardiographic findings, rheumatic heart disease and hypertension. One had definite signs of coronary-artery disease, and another a severe congenital abnormality of the heart. In many patients, the increased blood pressure went down when agitation subsided as a result of electroshock therapy. In a few other cases, the blood pressure went up or up and down in a way that was not always consistent with the patient's emotional state.

Eight patients with active and 25 with arrested pulmonary tuberculosis were treated in this series. Shock therapy of mental patients who are suffering from tuberculosis carries definite risks, but in selected cases, electroshock therapy has to be given even if the tuberculosis is active. If there is a history of recent hemorrhage or if the activity is high, shock should be given only when the pulmonary process has been controlled by collapse therapy. In 1 case electroshock therapy and pneumothorax were applied simultaneously.

Patients suffering from acute infections should be excluded from electroshock treatment, and every effort should be made to discover intercurrent infections prior to and during a course of electroshock. This was emphasized by the one fatal case after electroshock therapy that occurred in this series.

Eleven cases of fractures due to electroshock treatment were observed; 2 of them occurred in patients who had bone disease prior to the treatment. All fractures healed without permanent damage. Regarding the preliminary administration of curare, the opinion is expressed that the danger of this drug outweighs its advantage.

A few patients with diseases of the central nervous system were treated without ill effects. Another patient, who had no abnormal physical findings prior to electroshock treatment, developed a

transient hemiplegia following the treatment, most likely on the basis of vascular spasm, causing reversible structural damage to parts of the brain.

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MEDICAL PROGRESS

X-RAY DIAGNOSIS OF PULMONARY LESIONS

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THE roentgenologic examination of the chest readily reveals abnormality, but the determination of the exact nature of the process is more difficult. One way of approaching the diagnosis is dependent upon a knowledge of gross anatomy, pathology, physiology and x-ray shadows, in addition to having available the brief facts concerning the clinical status of the patient. The material collected for this review is presented on this basis.

From the anatomic standpoint, the recent studies of Boyden and his associates[1–3] of the bronchial and vascular structures of the lung, particularly the relation of the various segments, have been of value. Roentgenologically, Robbins et al.[4–10] have demonstrated the segments as they appear in collapse and the configuration of certain anatomic structures, such as the vascular network and hilar shadows and the interlobar septums or fissures; these have been found to be of importance in diagnosis. Medlar,[11] in a study of 1200 pairs of lungs, found that on the left side the major fissure was complete in 82 per cent, whereas on the right it was complete in approximately 70 per cent, and the minor fissure was complete in 38 per cent; in the other cases, the fissure was partially present. (Whether the fissure is complete or not is of relatively little importance in determining its position roentgenologically in the majority of patients.)

The azygos septum is important only in that it should not be confused with an abnormal process. Its incidence, as pointed out by Etter,[12] is 1:384 chests examined; Pendley[13] found it to be 1.6 per cent of 25,000 fluororoentgenograms.

Lachman[14] reviewed present-day teaching of anatomy from a critical standpoint, stressing the fact that the dynamic aspects as disclosed by x-ray examination differ considerably from the anatomy of the cadaver. Anatomy is not a fixed study, but one in which there is a good deal of motion of most of the thoracic structures. The posterior reflections of the pleura, sometimes spoken of as the pulmopleural demarcation lines,[15] are of diagnostic importance; loss of visibility of these lines in the roentgenogram may signify enlargement of the heart, pulmonary disease or fluid in the pleural space.

Westermark[16] has utilized changes in intra-alveolar pressure in diagnosis of pulmonary as well as mitral lesions. He has been able to show that the degree of filling of the pulmonary vessels and of the chambers of the heart is profoundly affected by intra-alveolar pressures. A water manometer with a tube held in the patient's mouth is used so that pressures of water from −15 up to +80 cm. are obtained. This method can also supply a rough measurement of the pressure in the pulmonary arteries by determining at which level vessels in the hilus diminish in size.

Technic of Examination

From the technical point of view, there is still a tendency for utilization of the black and white films showing marked contrast. This technic is rather attractive, but unless additional procedures, such as the Bucky or grid films or more complicated methods, are used, the information that might be obtained by means of the roentgen-ray examination is considerably curtailed. For those who desire as many data as possible from conventional films, the optimum kilovoltage technic, as suggested by Fuchs,[17] will yield a far greater return. This technic produces a film that is rather gray, but all portions of the chest are adequately penetrated so that more

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structures are visualized on the film than is possible with films of greater contrast. Although the lateral view has long been stressed as necessary, it is still too often neglected. If one is to visualize satisfactorily the septums and locate the various segmental lesions, it is vital to have a lateral projection.[4,5] Fleischner[18] has recently called attention to the value of the visible bronchial tree on conventional films as indicating pneumonic and other pulmonary consolidations — in other words, if the bronchi are visible, it is indicative of consolidation or a similar process within the adjacent lung, and their visibility distinguishes the abnormality from a pleural effusion or thickened pleura. Infrapulmonary collections of fluid may be readily confused with an elevated diaphragm,[19,20] and it may be necessary to examine the patient by means of films taken in the lateral decubitus position or after pneumoperitoneum or pneumothorax.

Numerous ways of obtaining satisfactory bronchograms,[21–23] including aspiration, catheter injection and injection under bronchoscopic control,[24] are available. The most important factor from the radiologic standpoint is that the major bronchi of all the lobes be filled in the average case, or that selected bronchi be filled in the special case. The position of the patient during the flow of the oil is of extreme importance. Kooperstein and Bass[25] call attention to 2 cases of bilateral pneumonia following instillation of lipiodol; this complication, however, is unusual.

Laminagraphy, planigraphy and tomography have received a great deal of emphasis in the past few years.[26–29] These procedures are of value in cases in which the patient is not to be bronchoscoped. They may help in demonstrating narrowing or obstruction of the major bronchi, in differentiating enlarged lymph nodes from hilar vascular structures and in disclosing certain cavities. In some cases peripheral masses, when they are benign, may be shown to displace bronchi and vessels, whereas malignant lesions do not displace the bronchi and if they invade the bronchi the latter are narrowed or occluded. Often, the actual tumor within the trachea or bronchus can be clearly delineated, but such a tumor will not be distinguishable from any other mass present within these structures. Laminagraphy can frequently replace bronchography and bronchoscopy when these procedures are impossible; the usual projections, including posteroanterior and lateral, can be used. Amisano[30] utilized transverse laminagraphy of the chest. The patient is in a sitting position with the head flexed on the chest and the film is under the thighs; the axis of rotation of the laminagraph is at the level of the chest to be examined. Considering the long object-film distance the laminagraphs reproduced were surprisingly good.

A recent suggestion for better delineation of chronic draining empyema cavities is mentioned although they are seen less frequently today than in the past; this is the use of a solution of 50 per cent pantopaque (iodophenylundecylate).[31]

Despite all the advances in technical methods, there still remains a large group of lesions that are not yet readily visualized on the roentgenogram. It has been our experience, confirmed by Ritter and Pendergrass,[32] that many of the small lesions of tuberculosis and metastatic nodules even as large as 1 cm. in diameter, particularly if they lie against the pleura, may be invisible on the x-ray film.

Mass chest surveys have proved valuable in the discovery of unrecognized cases of tuberculosis and other processes within the chest. A fair trial of photofluorography was given during World War II and is being continued at the present time. Numerous reviews stressing the value of these routine screening methods have been published; approximately 2 to 9 per cent of the films have disclosed pulmonary abnormalities, as many as 5 per cent of which have been found to be due to reinfection pulmonary tuberculosis. Repeated surveys in industry and elsewhere have shown that active pulmonary tuberculosis developed as a rule at the expected yearly rate; in suspicious cases, serial observations often confirmed the belief that active minimal tuberculosis is asymptomatic and can be discovered only by radiography of the chest.[33–40]

It should be borne in mind that photofluorography is a purely survey procedure rather than one that has accurate diagnostic value. Even with close attention to the use of proper developers, correct type of film and screen to provide excellent contrast and detail, these surveys cannot yield the information furnished by a complete roentgenologic examination of the chest. Another fact to be remembered is that this form of study will pick up many lesions for which the proper method of treatment may constitute a difficult problem for the internist, the radiologist and the surgeon.

Diagnosis of Inflammatory Lesions

Myers,[41] in a comprehensive article, reviewed the experience of twenty-six years' observation in the city of Minneapolis. The results demonstrated that carefully planned, long-term studies must be undertaken to evaluate such a chronic disease as tuberculosis. As far as the development of radiologic procedures is concerned, the paper films used at first have been succeeded by photofluorographic methods. In the early years there was considerable discrepancy between the findings on x-ray examination and those on tuberculin testing. After ten years, it was found that x-ray inspection of the chest rarely revealed evidence of significant disease in children under twelve or fourteen years of age even though they reacted positively to tuberculin; as a

result, routine roentgenologic studies of this age group were discontinued. After 1931, the use of stereoscopic films was also dropped since nothing significant could be detected on one film of a stereoscopic pair that was not equally discernible on the other. X-ray examination of nonreactors was found to be noncontributive, and it too was discontinued. Myers stressed the fact that children showing positive tuberculin reactions should be followed for many years with careful checkups. He recommended that no one attempt to make a conclusive diagnosis by an x-ray shadow and decried the possibility of determining the activity of a tuberculous lesion by a single x-ray examination.

It is of interest to note that in Minneapolis the tuberculin test is used as the method of screening in preference to x-ray examination, whereas in China it has been found that tuberculosis is so prevalent that x-ray films are preferred to the tuberculin test.[42]

Reinfection tuberculosis does not always make its first appearance in the upper lobes. Cherry[43] reports that in 1379 known cases of reinfection 1.52 per cent were discovered to have had a basal onset of the disease. In a survey of 500 cases with skeletal tuberculosis, Mann[44] found that 57 per cent had active pulmonary tuberculosis. He concluded that the skeletal lesion in most children originated soon after the primary infection, whereas in adults it was usually due to reinfection of a previously quiescent focus, which resulted in blood-stream dissemination and subsequent implantation in the bone and lung. Flaxman[45] advocates the use of a modified posterior lordotic projection, sometimes spoken of as the "apical view," for early recognition of tuberculosis in the apical portions of the upper lobes. This position enhances the visualization of the apexes by displacing the shadows of the clavicles away from the area to be studied. Medlar,[46] in an anatomic investigation of lungs of persons who had died unexpectedly, reports that apical scars were present with equal frequenccy in those who exhibited no microscopical evidence of pulmonary tuberculosis and in those with single and multiple foci. He believes that apical caps are not necessarily related etiologically to tuberculous infection. In tuberculosis, apical scars occur rarely in persons under the age of thirty; over the age of fifty, they are quite frequently due to tuberculosis, and an inequality of these shadows on the two sides should suggest the possibility of a tuberculous lesion.

That bronchial occlusion often accompanies tuberculosis is stressed in at least two reports. Hutchison[47] believes that the extensive shadows seen in primary tuberculosis are due to collapse and do not indicate pneumonic consolidation. Occlusion of the bronchus is most often caused by enlarged lymph nodes in association with sticky mucus from a swollen hyperemic mucous membrane. Guggenheim,[48] investigating pulmonary emphysema and tuberculosis, comes to the conclusion that in only a small minority of the cases can changes be explained on a purely compensatory basis. Localized emphysema, blebs and bullae are due to bronchial changes; the obstruction of the bronchi and bronchioles is caused by exudate, necrotic material, endobronchial tuberculosis, compression, torsion and stretching.

Considerable literature is accumulating that tends to indicate that many of the multiple areas of calcification seen within the lungs are secondary to histoplasmosis rather than to tuberculosis.[49–51] This conclusion is based on a negative tuberculin reaction and a positive reaction to histoplasmin. It is thought that histoplasmosis has a tendency to show the following features: the presence of multiple lesions in the parenchyma; the rather frequent occurrence before the age of one year; and progression of the pneumonic infiltration with the development of new lesions and appearance of calcification over a period of years. It is believed that a far greater number of children, particularly in the Middle West, are sensitive to histoplasmin than to tuberculin. Holt[52] reported the radiologic appearance of the chest in 5 autopsied cases of fatal histoplasmosis. These findings were exceedingly variable: 1 case showed miliary parenchymal lesions with hilar adenopathy; in another, patchy pneumonitis was present; in a third, a large area of consolidation was found in the right upper lobe; a fourth case showed rather widespread dissemination of miliary lesions; and the fifth disclosed extensive consolidation of the right lung. These findings indicate that there is no characteristic roentgenologic appearance to be expected in active histoplasmosis.

Torulosis,[53] aspergillosis,[54] nocardia asteroides infection[55] and the pulmonary manifestations of amebiasis[56] have no typical appearance and are often confused with tuberculosis (or tumor). Another more common chest condition, likewise difficult to distinguish from tuberculosis, is pulmonary coccidioidomycosis.[57–59] Although it has been considered more or less endemic in the Southwest and West, numerous cases of the infection are being found throughout the country in former members of the armed forces. Many of the initial infections are asymptomatic. In symptomatic cases, it has been noted that the onset is often abrupt, resembling an acute respiratory infection. The early roentgenologic picture may be similar to that of an atypical or bacterial pneumonia; the process may be lobular or lobar, and enlarged hilar lymph nodes may be present. Occasionally, pleural effusion may accompany and obscure the underlying process. Nodular densities may appear that ultimately break down and form cavities. Resolution, which may take place in from one to three weeks, is some-

times complete, leaving no scar; at other times, there is healing with fibrosis. In the disseminated form, the roentgenologic appearance may resemble other fungous infections, sarcoidosis, miliary tuberculosis or lymphatic spread of metastatic cancer throughout the lungs. The most common error in differentiation is with tuberculosis.

Boyer[60] reported 50 unselected cases of pulmonary tuberculosis in which bronchoscopy was done. Bronchiectasis was demonstrated in 85 per cent; in some cases it had not been suspected clinically. Collapse treatment seemed to bear no relation to the incidence of bronchial dilatation, and the degree of bronchiectasis had no apparent bearing on the amount of sputum.

Some interesting observations on the occurrence of bronchiectasis that was either associated with or the result of a primary tuberculosis were made by Jones and his associates.[61] In 85 of 716 children with pulmonary tuberculosis, a dense homogeneous, and usually segmental or lobar, shadow suggested the presence of an obstructive pneumonitis. Forty-two of the 85 patients were examined bronchoscopically, and 31 were found to have tuberculous involvement of a bronchus in the form of ulceration, granulation tissue, tuberculoma or extrinsic pressure from enlarged lymph nodes. Several years later some of the same children, together with others showing the same phenomena, were studied by bronchography. In 34 of the 37 children examined satisfactory bronchograms were obtained, and in 24 of these bronchiectasis was evident; the pulmonary lesion had been present twelve months or less in 4 of the latter and for thirteen months or longer in the other 20. The bronchographic examination was done at a mean interval of three and a half years after roentgenologic evidence of clearing of the original lesion. From these observations, it was concluded that the duration of the disease was important in the development of bronchial dilatation and that the damage was permanent. There was a preference for localization of the bronchiectasis to the anterolateral branch of the upper-lobe bronchus and the apical branch of the lower-lobe bronchus. The children had symptoms representing both the primary complex and bronchiectasis, but after clearing of the pneumonitis and tuberculosis, there were few symptoms of bronchiectasis.

Bronchiectasis as an entity has received a large amount of study and comment in the past, and since many cases are surgically curable it is desirable that the diagnosis be made promptly. It is the opinion of many that bronchograms are a necessity in making the diagnosis,[62] whereas Good[63] points out that in 75 per cent of the cases it is possible to make a tentative diagnosis of bronchiectasis from the plain films. He cautions also that it may be suspected more frequently from the routine films than can be confirmed by bronchography. Certain plain films will show no evidence of bronchiectasis even though the disease is actually present, and Good believes that bronchography is necessary in patients who give clinical evidence of it even though no lesion is demonstrable on the plain films. (Usually, it is best that bronchography be instituted at the request of the surgeon who is to perform the operation, inasmuch as the diagnosis will probably have been established by the plain films and the chief object of bronchography will be to furnish the surgeon with an adequate map of the bronchial tree from which he can plan his attack. This procedure also gives him the most accurate means of follow-up studies of the patient in the years to come. Bronchography, performed merely as a diagnostic method without intent of immediate operation, may leave considerable residua within the lungs, which will interfere with follow-up examination.)

Although there is no intention of entering the controversy regarding whether atypical pneumonia causes bronchiectasis, reversible or irreversible, it is to be noted that some cases of bronchiectasis are first discovered during or after an attack of atypical pneumonia.[64] In cases in which the question whether bronchiectasis is associated with the persistence of a pneumonic infiltration arises, it is probably advisable to do bronchography and to follow it with a second bronchogram after a suitable length of time. Royce and Victor[65] believe that an interval of one to three months is sufficient length of time in which to determine the reversibility of the process. Spain and Lester[66] report a case in which bronchiectasis developed distal to a foreign body in a bronchus within a period of seven weeks. It is their belief that early destruction of the musculoelastic layer precludes the possibility of reversibility.

It is of considerable interest that the radiologist sees relatively few cases of lobar pneumonia due to the pneumococcus in this era of antibiotics. Apparently, most of these patients are being treated in the home and are not hospitalized to any extent. As far as diagnosis of lobar pneumonia or of other inflammatory conditions is concerned, it is important that the physician follow the patient until the lesion has cleared roentgenologically. It is surprising how many chronic diseases such as bronchiectasis and numerous tumors are first diagnosed as lobar pneumonia or some other inflammatory condition; later, as the symptoms become more severe, these diagnoses must be discarded.

That the roentgenologic appearance of atypical pneumonia may be somewhat bizarre and easily confused with other conditions is a well known fact. Atypical pneumonia may produce collapse rather than consolidation as demonstrated on the roentgenogram; it can cause lesions that involve the greater portion of an entire lobe, one or more segments of a lobe or a diffuse miliary process

throughout the lung fields. In the last type, the patient is usually seriously ill, and in some cases this diffuse mottling may indicate the beginning of resolution in a process that had shown widespread consolidation. The lateral view is essential to determine adequately the segments involved as well as the amount of atelectasis that is often associated with the disease.[67–70] The pulmonary type of tularemia and Q fever may produce small areas of pneumonitis, which roentgenologically may be confused with pneumonia.[71–75]

A condition that has received renewed interest in the past few years is Loeffler's syndrome.[76–79] The roentgenologic appearance is thought to be due to areas of infiltration or edema that are transitory and tend to migrate to various parts of the lung. No particular lobe is predominantly involved, although when the upper lobe is the seat of the process it may be mistaken for tuberculosis. Clinically, the patients are usually not very ill; a marked eosinophilia is present, and the sputum may contain eosinophils. Loeffler's syndrome may be associated with infestation by *Ascaris lumbricoides*, *Necator americanus* and *Endamoeba histolytica*, strongyloidiasis, allergies, chronic brucellosis and other diseases. As seen in the tropics, the appearance from the roentgenologic standpoint is frequently unique in that it produces a diffuse miliary appearance, which is of transient duration.

Harvey[80] reports 2 cases of clinically proved brucellosis that showed a tendency to multiple small nodular lesions throughout the lung fields. One patient was known to have had previous bilateral hilar-lymph-node enlargement. The radiologic appearance of the chest is very similar to that usually seen in sarcoidosis. McCort et al.[81] describe the findings in the latter condition. Enlargement of the paratracheal lymph nodes was found in all of the 28 cases comprising their series, with enlargement of the peribronchial lymph nodes in 25; pulmonary or parenchymal involvement was evident in 15. In 2 cases there was pleural effusion, and in 1, pericardial effusion; osseous changes were noted in the hands of 6 patients.

Numerous causes of emphysema and pulmonary fibrosis are known. In a recent symposium on so-called idiopathic fibrosis and emphysema, Mallory[82] has shown certain granulomatous or sarcoid-like lesions to be the inciting factor in a small group. The roentgenologic appearance of these cases differs considerably from the usual sarcoid findings; the markings are greatly thickened throughout the lungs, with large areas of intervening rarefaction, apparently representing emphysematous blebs or bullae.[83] Cases of idiopathic fibrosis run a rather severe course with a fatal termination.[84]

Acute pulmonary edema, a frequent accompaniment of acute nephritis, mitral stenosis and certain other conditions, produces fairly characteristic roentgen-ray shadows. In spite of the minimal signs and symptoms noted clinically, the radiographs usually show massive centrally distributed shadows in the lungs and a somewhat butterfly-like appearance. Doniach[85] ascribes uremic edema of the lungs to capillary permeability and says that it may be seen in other conditions causing heart failure. According to Burford and Burbank,[86] pulmonary edema in some cases is caused by blast injury in which a wave of highly compressed air produces severe changes in the parenchyma, the pleura and even the chest wall. Under these circumstances, the edema in all likelihood is traumatic and not the result of heart failure.

The appearance and technic of demonstration of pulmonary infarcts has been reviewed by Robbins.[87] An interesting but small group of cases are those in which there is infarction of an entire lobe such as that discussed by Rawson and Cocke.[88] Their patient, a thirty-nine-year-old Negro, had complete infarction of the left lower lobe subsequent to long-continued passive congestion; there was aseptic softening, with subsequent rupture into the pleural cavity resulting in hemopneumothorax. They found only 5 other cases of this type previously reported, and it is of interest that all the patients were between twenty and forty-four years of age.

Pulmonary Lesions in Children

An excellent approach to the subject of pediatric pulmonary roentgenology has been presented by Blair.[89] He summarizes and classifies in a concise manner the numerous conditions that may be encountered in children.

The surgical aspects of staphylococcal pneumonia in infancy and childhood have been discussed by Blumenthal and Neuhof.[90] The roentgenologic appearance is that of ordinary suppurative pneumonia, with densities that may be lobular, lobar or segmental in type and with a tendency to cavitation and abscess formation; there may be extension to the pleura. The authors believe that repeated roentgenologic examination is essential in following the course of the disease.

Another form of cavity, termed pneumatocele, presents a thin margin and may contain fluid. It is seen in the course of other types of pneumonia in children and has recently been re-emphasized by Almklov and Hatoff.[91] The pneumatocele develops in an area of consolidation; the time of its appearance, in the authors' series, varied from the second to the twenty-fifth day after onset of the disease. In certain cases more than one pneumatocele were present; these ranged in diameter from 1 to 3 cm., and fluid levels were frequently noted. The pneumatoceles disappeared spontaneously without treatment.

Of particular interest to the radiologist may be the child who is brought for examination of the chest with the question, because of his cyanosis, of pneumonia or heart disease. Recent reports[92, 93]

describe such a condition, which has been found to be due to poisoning from wax crayons. These children exhibit rapid respirations, some elevation of temperature and pulse, weakness and listlessness. The striking roentgenologic feature is the apparently normal chest and the finding of opaque material in the stomach and intestinal tract.

Idiopathic pulmonary hemosiderosis or essential brown induration of the lungs has been described by Wyllie et al.[94] All the patients were children ranging in age from a few months to sixteen years, with the exception of 1 man of thirty-eight years. The disease is characterized by recurrent attacks, sudden in onset, of fatigue, cyanosis and pallor, with increasing dyspnea, acceleration of the pulse rate and a high fever. Cough is troublesome, often followed by vomiting, and at times traces or even considerable quantities of blood are noticeable in sputum or vomitus. There may be abdominal pain as well as increased liver dullness and splenomegaly. The most common roentgenologic findings — and these become accentuated during an attack — are mottled shadows, particularly noticeable in the hilar areas, and a diffuse stippling throughout the lung fields. There may be evidence of partial lobar collapse. At first glance the miliary nodules suggest tuberculosis, but close inspection is said to reveal small, clear, circular spaces surrounded by thickened opaque walls, giving a pumice-stone appearance; the heart shadow is enlarged, the border extending chiefly to the right, and the pulmonary conus is prominent. The prognosis is poor.

A somewhat similar type of nodular lesion may be associated with mitral stenosis, and in long-standing cases actual calcification or ossification of some of these nodules may occur.[95]

Occupational Diseases

Occupational diseases that cause changes within the chest are numerous. Some of those reported recently have been associated with welding and inhalation of graphite dust and of dust caused by the handling of grains and seeds.[96–101] Bagasse disease of the lungs, or bagassosis, is brought about by the inhalation of dried bagasse dust. There seems to be an insufficient amount of silica present in the dust to cause the signs and symptoms.[102, 103] The severity of the disease appears to be directly related to the concentration and duration of exposure to the dust. Some cases end fatally. The roentgenographic appearance differs from that in other forms of pneumoconiosis primarily in that it is reversible, and resolution may be complete in six to eight months.

Exposure to beryllium compounds in industry has attracted considerable attention in the last few years.[104–108] There appear to be two forms of reaction to exposure, the first of which is an acute chemical pneumonitis resulting from exposure to high concentrations of beryllium compounds including fluoride, oxyfluoride and oxides. There is a definite relation between exposure and the development of the disease, and if the patient recovers, the symptoms clear up with elimination of the exposure. The second form is a delayed chemical pneumonitis, apparently caused by exposure to small amounts of beryllium compounds in the air. This is characterized by delay, which may vary from one month to five years, between exposure and onset of symptoms. The x-ray appearance, as described by Wilson,[109] is very similar to that seen in sarcoidosis.

Tumors

In the present era of thoracic surgery, the roentgenologic diagnosis of tumor of the lung, in an attempt to differentiate benign and malignant lesions, has become primarily of academic interest.[110–113] Bloch et al.,[114] pointing out the difficulties in differential diagnosis of bronchiogenic carcinoma, observe that diagnosis is often impossible by conservative methods and that surgical exploration is indicated. Churchill[115] states that the problem in primary cancer of the lung is the same as that encountered in any other form of cancer — in other words, earlier diagnosis. In his opinion, roentgenologic screening is at present the only way to detect early primary cancer in the apparently well population. This method will disclose silent neoplasms in the periphery of the lung, and in recent years it has succeeded in bringing to light a few asymptomatic cases. It will not, however, disclose the early endobronchial growth that has not yet encroached upon the lumen of the bronchus; these lesions are less likely to be silent, the patient having symptoms at an earlier stage than the one with a peripheral lesion.

Rigler and Kelby[116] have re-emphasized the fact that obstructive emphysema is a valuable early sign of bronchiogenic cancer. This is best demonstrated by roentgenograms during expiration or by fluoroscopy. Transition from emphysema to atelectasis occurs as the tumor grows and completely obstructs the bronchus. Reitz[117] and Oechsli and Olson[118] stress the necessity of adequate x-ray examination as well as the importance of photofluorography. In their opinion, laminagraphy is of definite value when the lesion is beyond the reach of the bronchoscope.

Gerstl and his colleagues[119] discuss 7 cases of coexisting bronchiogenic carcinoma and pulmonary tuberculosis, noting that there was little difficulty in making the diagnosis of tuberculosis but much in that of carcinoma. The x-ray examination was of the greatest assistance in raising the question of cancer. Hilar enlargement or infiltration, evidence of major bronchial obstruction, or a relatively circumscribed mass should, in the authors' experience, arouse the suspicion of carcinoma.

Lymphatic spread of carcinoma throughout the lungs may produce an appearance that is difficult to differentiate from sarcoidosis, silicosis and other miliary lesions. Clinically it is characterized by severe, increasing dyspnea in a patient with recognized or unrecognized carcinoma elsewhere.[120, 121]

Mediastinal Tumors

Numerous tumors of the mediastinum are found incidentally on pre-employment or survey examinations. Frequently cases of myasthenia gravis are found to be associated with thymic tumors, and any patient with this disease should have a thorough roentgenologic study of the chest. Bronchiogenic cysts and cysts arising from the gastrointestinal tract are often observed; these cannot readily be differentiated from malignant teratomas, neurofibrosarcomas and other malignant tumors.[122–127] In the differential diagnosis, one should exclude the possibility of aneurysm of the aorta, and this may be done by angiocardiographic visualization of the heart and great vessels.[128] An intrathoracic meningocele must also be distinguished from neurogenic tumors; this may be accomplished by myelography or possibly by the utilization of the Valsalva test during fluoroscopy.[129] Holt[130] called attention to right-sided epicardial fat pad as a differential point; these may be found in the right anterior costophrenic angle. Often, however, fat pads cannot be distinguished from cystic lesions, omental hernias or tumors in this location. The facts that malignant changes cannot be excluded in any mediastinal tumor and that there may be later infection of cystic lesions make it imperative that surgical removal be attempted whenever possible.

Summary

Methods for the roentgenologic diagnosis of pulmonary lesions are steadily improving. There remain, however, a large number of unsolved problems that offer a wide field for future investigation.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

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BENJAMIN CASTLEMAN, M.D., *Associate Editor*

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CASE 34471

PRESENTATION OF CASE

First admission. A seventy-one-year-old woman was admitted to the hospital because of the presence of an abdominal mass.

Eight years before admission the patient noted a walnut-sized mass in the right lower quadrant, which gradually increased in size. Six months before admission she first noted pain at the upper portion of the mass. There was no fever, chills, nausea, melena, night sweats or urinary symptoms.

Physical examination showed some tenderness in the right lower quadrant. A mass was palpable extending laterally into the right flank, and it was felt extending on to the crest of the ilium.

Laboratory examination revealed a negative urine, a white-cell count of 8000 and a hemoglobin of 13.5 gm. A barium enema showed diverticulosis of the sigmoid and upward displacement of the cecum by a large soft-tissue mass in the right lower quadrant. The terminal ileum lay along the medial and inferior margins of the mass.

At operation there was an encapsulated tumor attached to the ureter, bladder, appendix and the large vessels running to the right leg. In removing the tumor the right common iliac artery gave way and it was necessary to ligate it. Following operation she was treated with anticoagulants and exercises, and the leg remained viable. The pathological report on the tumor was fibroma, probably neurogenic.

Second admission (two years later). The patient remained well until three and a half months before admission, when she suffered an attack of "grippe." She did not feel that she regained her full strength following this illness and one month later she noted a yellow color to her skin. This was not associated with an acute episode of pain or nausea and vomiting. She did notice postprandial distress associated with belching, a feeling of fullness and bloating. The stools were light, and the urine dark, and there was generalized itching. In the two weeks prior to admission the jaundice lessened in degree, and the stools became darker and the urine lighter. Bilateral ankle swelling was noted.

On examination the blood pressure was 130 systolic, 80 diastolic. The main finding was an abdominal mass with an irregular edge, occupying the right upper quadrant, extending 15 cm. below the costal margin. The skin was icteric, and there was ++ edema up to the knees. The temperature was normal.

Laboratory examination revealed a urine specific gravity of 1.017, a + test for albumin and a +++ test for bile, and microscopically there was an occasional white cell and granular cast in the sediment. The white-cell count was 8600, and the hemoglobin 12.5 gm., with a normal differential. The van den Bergh test was 10.3 mg. per 100 cc. direct and 13.2 mg. indirect; the prothrombin time was 17 seconds (normal, 16 seconds), and a cephalin flocculation test was ++ at the end of twenty-four and forty-eight hours. The total protein was 6.72 gm., the albumin 3.7 gm., and the globulin 3.01 gm. per 100 cc., and the alkaline phosphatase was 24.6 units. A barium enema showed displacement inferiorly of the transverse colon and cecum by a large mass believed to be liver. A gastric series revealed displacement of the stomach and duodenum to the left by a mass in the right upper abdomen. A chest radiogram disclosed a rounded area of increased density, 1.5 cm. in diameter, in the left lower lobe, not present two years previously. An intravenous pyelogram demonstrated good function in both kidneys. A retrograde pyelogram showed a normal kidney on the right.

On the eighteenth hospital day a laparotomy was performed.

DIFFERENTIAL DIAGNOSIS

DR. CLIFFORD C. FRANSEEN: May we see the x-ray films?

DR. JAMES J. MCCORT: A plain film of the abdomen made on the first admission discloses a large, soft-tissue mass in the right lower quadrant. A barium enema shows diverticula in the sigmoid without signs of inflammation. In addition to that the cecum is elevated and a loop of terminal ileum displaced toward the midline, evidently around the mass. It does not appear to be intrinsic.

When the barium enema was done two years later the barium flowed readily from the rectum to the cecum. The diverticula previously described are filled at this time. However, a large mass is present in the right upper quadrant, displacing the transverse colon downward. Again, there is no evidence that the soft-tissue mass is intrinsic to the colon. A gastrointestinal series done on the second admission shows a suggestion of small varices in the lower third of the esophagus. The stomach is displaced to the left and slightly anteriorly, owing to extrinsic pressure. Studies were made to demon-

strate the right kidney and any possible relation of the mass to the kidney. On an intravenous pyelogram we see a normal outline of the kidney with good filling of the calyxes and pelvis and without distortion of these structures. Further study by the retrograde injection of dye in the right urinary tract confirms this. If we compare the films taken on the first and second admissions there is no difference in the appearance of the calyxes or in the configuration of the pelvis in two years. We can see the kidney in the later film behind the soft-tissue mass. It is a little lower in the abdomen than it was two years previously. The mass, therefore, is extrinsic to the right kidney and displaces it downward slightly.

There is a small, round area of increased density in the lower lobe of the left lung seen in the chest film taken on the second admission but not present on the chest film taken two years earlier. Otherwise the lungs are clear; the heart and vessels appear normal.

Dr. Franseen: Let us go back to the beginning and see what we can make of this tumor. It was evidently one of slow growth and when first noted was a walnut-sized mass in the right lower quadrant. We can tell something about the huge size of the abdomen from the x-ray films. If the patient was able to feel the mass herself through that abdominal wall, we can be sure that the walnut-sized mass was just a knob that protruded above a larger mass beneath. Eight years later she had a large lesion in the right lower quadrant consistent with slow growth. The slow growth was also suggested by the pathological report of a fibroma, possibly neurogenic. We get no exact idea of its site of origin from this record. It was apparently adjacent to or attached to numerous structures. With such soft-tissue tumors, one can cover oneself by saying that, from subsequent events, perhaps a section from another part of the tumor might have shown it to be more malignant than the histologic picture that was reported. In general, in dealing with tumors, if a secondary tumor appears, one should always assume that there was a relation with the first until proved otherwise. It may not have been so in this case. For two years she remained free of trouble. That is not an unreasonable interval for a tumor of this type even though there may have been a recurrence. Although the tumor was reportedly encapsulated, there was considerable difficulty in removing it — that is, it was not clearly encapsulated from a surgeon's viewpoint.

Two years later she had what was thought to be the "grippe," meaning, I suppose, that her family doctor had treated her for "intestinal grippe," and not necessarily an upper respiratory infection. The symptoms related to the stomach could be accounted for on the basis of pressure on the stomach or duodenum since the duodenum was pushed over to the right on x-ray study. There is no mention of blood in the stools. There is no way of knowing, of course, whether or not this was a second primary tumor, and this always has to be considered. I am assuming all along that this was a tumor. There is nothing to suggest that this was an inflammatory process from the history or the findings at that time, and I think it is safer to go on the assumption that this was a tumor until it has been ruled out, especially in a seventy-three year-old woman. If it turns out to be inflammatory, that is fine. There is nothing here to permit me to suspect that this was due to an inflammatory lesion of the biliary tract or to obstruction on the basis of stone in the common duct. One certainly has to consider that area in the lung as a metastasis and thereby strengthen the suspicion of tumor.

Could all this picture have come from one of the fibrocellular groups of tumors? Metastasis to the liver would be an unusual behavior for it, however. We assume that the palpable mass was the liver, but the mass could have been in the retroperitoneal structures and the lymph nodes, giving the same displacement on x-ray examination, but this would also be somewhat unusual for the type of tumor described, even the malignant forms. The same statement is true of the neurogenic group. In my experience these two often show up in the lung but rarely in the liver. If we dismiss that, we will have to cover ourselves further by saying that it may have been some other form of malignant tumor, possibly a leiomyosarcoma. It would fit in, but not well. So, taking everything into consideration, I think it is safe to assume that a second primary tumor would not be uncommon, especially in a seventy-three-year-old woman.

What primary tumor can cause these findings? We have not much evidence for a malignant lesion anywhere else in the gastrointestinal tract. The jaundice could well have been due to pressure at the hilus of the liver on the bile duct. The only thing I can see against it is that the findings suggest that the jaundice lessened. Almost invariably with malignant tumors jaundice is progressive and becomes complete rather than being intermittent when it is caused by obstruction from a stone. That is the only finding against this idea, however. We must consider other things of course. The whole abdomen is open to suspicion — the gastrointestinal tract and the ovaries, for example. I think we can assume that the breasts and other peripheral sources of origin have been ruled out, or some mention would have been made of them. We assume perhaps that the left kidney was normal. The retrograde pyelogram was done only on the right. Is that correct?

Dr. McCort: Yes.

Dr. Franseen: There was no question that function was good in the left kidney. There is nothing

to suggest any abnormal condition in the stomach. All the x-ray findings suggest pressure extrinsically. If one comes down to an eventual analysis and is forced to choose any particular tumor, one might say that this was a malignant tumor of the fibrocellular group, arising in the retroperitoneal structures, with metastases to the lung, but the metastasis to the liver still bothers me. The picture, to my mind, would better fit something like a new primary tumor in the liver, which could of course go to the lung like this and form this large mass. Nothing is said about ascites. I think we come down to the point where the diagnosis might be academic. One can only prove it by histologic examination. This answer might have been obtained by a peritoneoscopy. Presumably a laparotomy was done with the idea that if the gall bladder was not involved, a short-circuiting operation could be done, and the patient could be given some temporary relief from the jaundice. That is as far as I can go toward establishing the histologic diagnosis, except to say that the retroperitoneal tumor had apparently involved the liver. If I were to make a choice preoperatively, I would list a primary hepatoma of the liver.

Dr. Wyman Richardson: There is nothing suggested from the history that there was underlying disease of the liver. Usually the primary hepatoma develops on top of it.

Dr. Franseen: That is true. The only clue here is the suggestion of esophageal varices.

Dr. Fred A. Simmons: I operated on this patient at the first admission. She will always remain a graphic case because of the accident to the wall of the internal iliac artery, which was of fairly good size. The tumor encircled the great vessels behind as well as on top. The ureter lay in the curve of the lateral aspect. As the tumor was dissected from the common iliac artery the vessel separated apparently at the site of a calcareous deposit. We altered our technic and the tumor was freed after the hemorrhage had been controlled and transfusion started. The patient's condition was good enough to allow removal of the tumor, though we thought we had removed it in its entirety. I am impressed with the careful postoperative handling by the house staff, which enabled this woman to keep the extremity. The pathological report was a benign tumor, but my impression at the operation was that it should have been malignant. I did not palpate the liver, since we approached the growth retroperitoneally.

Clinical Diagnosis

Metastatic fibrosarcoma of liver.

Dr. Franseen's Diagnosis

Hepatoma of liver.

Anatomical Diagnosis

Metastatic fibrosarcoma of liver and lungs.

Pathological Discussion

Dr. Benjamin Castleman: When the patient came back the second time the service asked us to review the slides of the original tumor, which we did, and felt again that from the block we had it looked like a benign fibroma. A peritoneoscopy was unsatisfactory, so that a laparotomy was done and a tumor found within the liver, a biopsy of which showed fibrosarcoma. The patient did not do well following that procedure and died.

At autopsy she had a liver that weighed almost 6 kg., being diffusely involved with round nodules

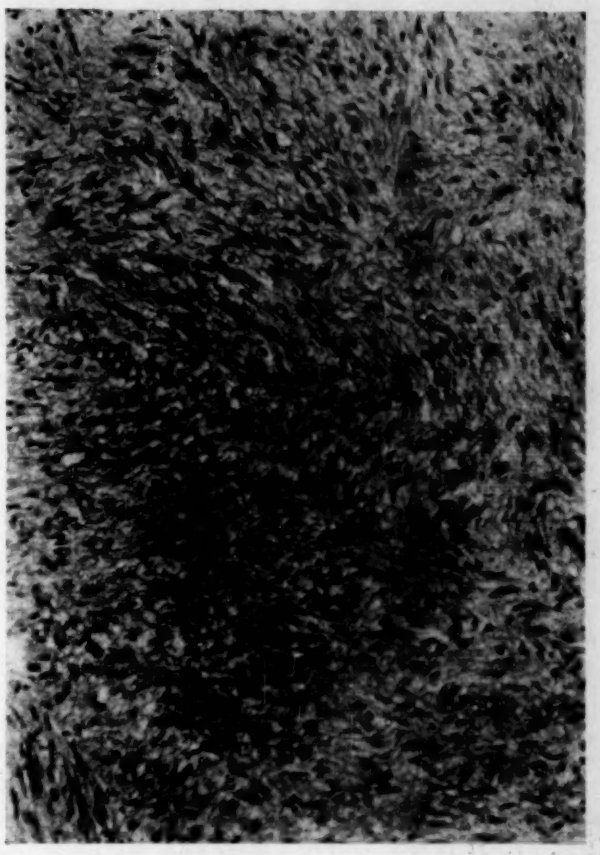

Figure 1.

of metastatic sarcoma. The metastases were very circumscribed and could easily be shelled out, a feature that distinguishes it from metastatic carcinoma. The jaundice was probably due in part to the neoplastic replacement of the liver, but I believe more likely due to the fact that the caudate lobe was completely involved with tumor and pressed against the common bile duct. There were two nodules in the lungs — one on the pleura of the left lobe and one within it.

DR. ALLEN G. BRAILEY: Why did the jaundice lessen?

DR. CASTLEMAN: Perhaps the caudate lobe flopped around a bit.

DR. JAMES T. HEYL: Was there anything left in the right lower quadrant?

DR. CASTLEMAN: The pelvis was completely free from tumor. The right ureter was thickened on the outside where the tumor apparently had been adherent but there was no evidence of tumor. We

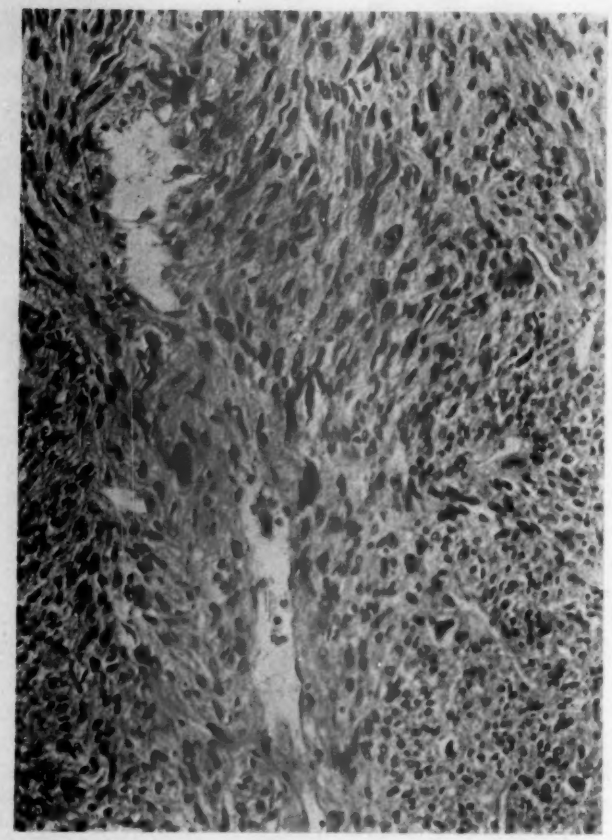

FIGURE 2.

took several sections from that area. Here is a photomicrograph of the original fibromatous tumor (Fig. 1). It is not very cellular, and the individual cells have single nuclei. In the section of the liver biopsy, on the other hand, the cells have changed. The nuclei are very large; some of the cells have two and three nuclei; and occasional mitoses are seen (Fig. 2). It is possible, as Dr. Franseen has mentioned, that if more blocks from the original tumor had been taken, we might have found one or two areas with morphologic evidence of malignancy.

DR. RICHARDSON: Is it possible that this was a new tumor?

DR. CASTLEMAN: I do not believe so. These certainly were metastases in the liver.

DR. FRANSEEN: Can you remember how many other times you have seen a fibrosarcoma behave this way?

DR. CASTLEMAN: I think we have had one other case. However, most of these cases that metastasize go to the lungs.

DR. SIMMONS: In retrospect, it would have been wise to radiate her.

DR. CASTLEMAN: No, I do not believe so. The metastases had probably already occurred at the time of the original operation.

DR. FRANSEEN: I also think that the metastases were there at the time of operation. Again we should emphasize the fact that hepatic metastases alone are not a contraindication to palliative surgery in certain carcinomas and other tumors—for example, cancer of the rectum. In this case it is possible that metastasis to the liver was not recognized at the original operation, and perhaps fortunately so, otherwise there might have been a temptation to abandon the difficult removal, and the patient would thereby have been denied the two years of palliative relief that she obtained.

CASE 34472

PRESENTATION OF CASE

A fifty-five-year-old rubber-factory worker was admitted to the hospital because of hematuria.

Approximately five months before entry he began to have intermittent hematuria, with blood clots. This was associated with a low backache and some dysuria. He had no pain in the flanks, loins or lower abdomen. He had nocturia four or five times a night but rarely any urgency. There was no weight loss.

Physical examination revealed a slim, emotionally unstable man who appeared younger than his stated age and who seemed in good physical condition. Aside from a moderately enlarged prostate gland, the physical examination was negative.

The temperature, pulse and respirations were normal. The blood pressure was 130 systolic, 90 diastolic.

Examination of the blood disclosed a white-cell count of 10,000 and a hemoglobin of 12.8 gm. The nonprotein nitrogen was 32 mg. per 100 cc. The urine showed a ++++ test for albumin, and the sediment contained 100 to 120 red cells and a rare white cell per high-power field. The specific gravity was 1.015. A urine culture grew *Staphylococcus albus* and alpha-hemolytic streptococcus.

Cystoscopy revealed a small, papillomatous growth, measuring approximately 1.5 cm. in diameter, just behind the interureteric ridge. Another round mass, measuring approximately 3 cm. in diameter and having what appeared to be a larger stalk, was noted just to the left of the

left ureteral orifice. No blood was seen coming from either orifice.

An intravenous pyelogram showed a filling defect measuring approximately 2 cm. in diameter in the left kidney pelvis and in the adjacent inferior major calyx (Fig. 1). In none of the films did the area fill, and one of the calyxes was deformed, suggesting the presence of a mass in that region. The lateral margin of the left kidney was not clearly visualized, but there was some suspicion of an increase in size extending outward from the lateral margin in its middle portion. The right kidney was normal in size, shape and position.

On the nineteenth hospital day an operation was performed.

Differential Diagnosis

Dr. Richard Chute: To begin with, the fact that this patient was a rubber-factory worker may have been of significance. Although we do not know how long he had been at this work or exactly what he did, the possibility of poisoning with benzol, with an aniline compound or with lead must always be considered in rubber-factory workers. Lead is used in the process of manufacture, benzol is widely used as a solvent, and aniline compounds are employed to accelerate vulcanization. In benzol poisoning the bone marrow is affected, with reduction in platelets and a tendency to abnormal bleeding from the gums, nose and so forth. Frequently there is leukopenia (although the white-cell count may be normal or may be elevated), and there is anemia. In this patient there was no mention of bleeding other than intermittent hematuria, consistent with his bladder tumors; his white-cell count was not remarkable, and there was very little anemia so I believe that benzol poisoning can be ruled out. Also, I feel that lead poisoning can be ruled out, since no colic, weakness, wrist drop, lead line and so forth were mentioned. When it comes to the question of aniline poisoning he was not described as cyanotic like the rubber-factory workers who used to be called "blue boys" in Akron, Ohio. Nevertheless, it is a fact that workers with aniline compounds have a well recognized abnormal tendency to the formation of bladder tumors, which are said to occur in them thirty-three times as frequently as among other people. The tumors usually do not appear until after prolonged exposure — the average time being twelve years. A doubtful privilege of working in plants where a good deal of aniline is used is that of receiving, at the company's expense, periodic cystoscopic examinations. Furthermore, long exposure to aniline is well known to make men irritable and grouchy, and this patient is described as "emotionally unstable." So it is possible that aniline poisoning played a part in this case.

The finding of two pedicled papillary tumors is not surprising in a fifty-five-year-old man with a history of bladder symptoms and the presence of blood in the urine. In my opinion these are undoubtedly papillary carcinomas, since I know of no other bladder disease that produces this picture. Bilharziasis (schistosomiasis) may produce a somewhat similar appearance, except that the lesions are more sessile and are usually ulcerated, and there are usually more marked symptoms and evidence of inflammation. The finding of only rare white cells in the urine indicates that the lesions in the bladder were not inflammatory. Nothing is said about residence in areas of schistosomiasis such as Egypt or of finding the ova of *Schistosoma haematobium* in the urine.

Figure 1. *Intravenous Pyelogram.*
Arrow points to crescentic calyx.

Next we come to consideration of the significance of the filling defect in the left kidney pelvis associated with deformity of a calyx, suggesting a mass. Naturally, from the x-ray reports one thinks at once of a malignant papillary tumor of the kidney pelvis that has invaded the kidney parenchyma. It is well known that papillary tumors of the kidney pelvis often invade the kidney, extend down the ureter and give secondary implants in the bladder. Perhaps the tumors in the bladder were not true implants. Perhaps they occurred simultaneously with the renal tumors, very possibly brought on by the same etiologic factor. At any rate, the association of papillary tumors of the kidney pelvis with papillary tumors of the bladder is a well recognized fact. It is believed that this double lesion is greater in persons exposed to aniline than in others.

The fact that at cystoscopy no blood was seen coming from either ureteral orifice does not rule out the kidneys as a source of bleeding in a case with intermittent hematuria. In this patient the

hematuria could just as well have come from a kidney as from the bladder tumors — very likely from both. Regarding the differential diagnosis of the x-ray picture, blood clot could have caused a filling defect in the kidney pelvis, but I would hardly expect it to deform a calyx or produce a picture suggestive of a mass. However, a tumor with blood clot could give this picture. Other tumors than papillary ones, such as invasive squamous-cell carcinoma of the renal pelvis and, rarely, renal-cell carcinoma (so-called hypernephroma), could also produce this appearance. Renal cyst is another possibility. This could cause deformity of a calyx and the suggestion of a mass, and, if it bled, as they sometimes do, the resulting blood clot could cause a filling defect in the kidney pelvis. Rarely, a staphylococcal abscess of the kidney parenchyma — the so-called renal carbuncle — gives this appearance on x-ray study. In the record no mention is made of the usual preceding history of recurrent furuncles or other infected skin lesions or a paronychia. Also, there was no fever and no mention of pain or tenderness in the left kidney region. The staphylococcus cultured from the urine was *Staph. albus* rather than *Staph. aureus*, the usual organism in these processes. Therefore, I do not believe that we are dealing with a renal carbuncle.

The final note says that "on the nineteenth hospital day an operation was performed." Since it is most unusual for the Urological Service to wait as long as this to operate, I suspect that there was something very unusual about this case. However, on the basis of the evidence submitted to me, I am making a diagnosis of carcinoma of the left kidney, probably papillary, and papillary carcinoma of the bladder.

Dr. Stanley M. Wyman: The plain film of the abdomen shows the kidneys fairly well outlined. I am unable to get very much excited about any distortion of the lateral margin of the left kidney. These first three films of the intravenous pyelogram examination are the most striking. There is a crescentic dye shadow overlying the lower major calyx. The appearance is that of compression and displacement of a minor calyx and compression of the pelvis of the kidney. The major calyxes, as far as we can see in the upper pole, appear normal. The lowermost calyx and the calyx extending to the left lower quadrant are not adequately seen. From this examination one can assume the presence of a mass, 2 or 3 cm. in diameter, compressing the calyxes in this region. The last film taken from the retrograde examination better demonstrates the lower major calyx. I can see no definite intrinsic involvement of this and there seems to be merely pressure on a minor calyx and distortion of the lower major calyx. Again there is possibly a little upward displacement of the major calyx in the midkidney region, giving the impression of something in this region compressing but not intrinsically involving the major calyx. I do not believe that the kidney pelvis per se is involved in this process. I think it is most likely a mass in the parenchyma rather than in the calyx.

Dr. Chute: That would throw out papillary carcinoma of the kidney and make a renal-cell carcinoma or cyst a better choice.

Clinical Diagnosis

Papillary carcinoma of kidney and bladder.

Dr. Chute's Diagnoses

Renal-cell carcinoma or cyst of kidney.
Papillary carcinoma of bladder.

Anatomical Diagnoses

Cyst of kidney.
Papillary carcinoma of bladder.

Pathological Discussion

Dr. Benjamin Castleman: The first operation was a biopsy of the tumor of the bladder. This proved to be papillary carcinoma, and following that operation the left kidney was removed. When we received the kidney we found a well encapsulated, previously sectioned cyst about 2 cm. in diameter in the midportion of the kidney, extending down and compressing the calyx to the lower pole of the kidney. The cyst had a very thin wall and apparently had been filled with clear, thin fluid. There was a thin layer of cortex over the cyst wall so that I do not believe it had produced much, if any, bulge of the margin of the kidney.

The patient worked in a factory where rubber products, such as raincoats and boots, are made and not in the manufacture of rubber. I should doubt that he was exposed to beta-naphthol, which is the substance in the aniline dyes that is responsible for the development of bladder cancer.

Dr. Arthur W. Allen: What did they do to the bladder tumor?

Dr. Castleman: It was resected by cautery just a few days ago.

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TO HIM THAT OVERCOMETH

A series of short papers have appeared this year in the London *Lancet* that are in no sense scientific or technical contributions; nor are they clinical, in that they do not describe from a professional point of view the symptomatology of the disorders discussed, or their treatment. Under the general heading "Disabilities" they consist merely of the patient's own brief chronicle of the disabling features of a disease or an injury, the method of meeting them and the degree of success attained in overcoming them.

The physical means by which some of these disabilities are defeated or their effects mitigated is of interest; the real worth of the articles lies in their inspirational value — the spiritual lesson that they teach of the strength that comes through the effort of resistance. Each instalment in the series is a chapter from the book of courage.

A girl of twenty-three with a crushed vertebra and a damaged spinal cord copes with the problem of urinary incontinence and forces herself to become again a useful and optimistic member of society. A young man with a lesion of the cauda equina teaches himself to overcome his own bowel and bladder difficulties and learns to walk again. An Englishwoman with disseminated sclerosis, uncoordinated to the point where she reels and staggers like a drunken person, is caught in the German occupation of France. Considered by the conquerors as incapable of resistance she organizes a factory for forging identification papers and, with one leg splinted, relearns bicycle riding. Alone she travels through the countryside, picking herself out of the ditch after each of her frequent catastrophes, finds fugitive English and American flyers and arranges for their escape. A victim of the crippling effects of her disease, she apparently finds also some measure of compensation in the emotional exaltation that sometimes accompanies it.

A man with double forearm amputation as a result of a bomb explosion employs to their fullest possibilities the varied mechanical contrivances available for amputees, as well as those that he has himself invented or improved. The best years of his life he finds to be still ahead of him. A patient with congenital cerebral palsy, compounded with infantile paralysis contracted at the age of two, considers life to be a "total war against physical affliction." "When told I cannot do a thing I have sometimes to sit and think out my own way of doing it, and then I make this way as much like the normal method as possible." It is this person, once "the peculiar child of all peculiar children," who becomes a lecturer and lay preacher, who teaches himself to ride a bicycle, to drive a car and to manage a horse.

Another victim of a bomb explosion, suddenly and totally blinded at the age of thirty, discovers that "one of the compensations of blindness is finding ways of overcoming it." It is this person who, having decided that such recreations as billiards

and table tennis are beyond his capacity, turns successfully to high diving, surf-riding and aquaplaning. For him the words of England's blind poet have had peculiar meaning:

> . . . who best
> Bear his milde yoak, they serve him best. . . .

And so the list has grown — the education of a congenitally deaf child as told by its parents, the adjustments made by a nurse who develops epilepsy at thirty, the problems of the tuberculous, the young man crippled by poliomyelitis, the adolescent boy with an ileostomy, the alcoholic who finds his salvation in helping others similarly afflicted.

Overcompensated many of these witnesses may be called, and with reason, as the heart hypertrophies that has an extra load to carry. From their achievements, however, a fundamental lesson may be drawn. All forms of life are meant to struggle against their difficulties and to rise above their obstacles or succumb. These that have been presented are only extreme examples culled from recent experiences.

If man, individually or in the aggregate, cannot accept life's challenges he must accept the futility of life. In this season set briefly apart for gratitude let him, then, rather than being thankful only for what apparent good fortune may seem to have overtaken him, be thankful also for the problems, the adversities, the temporary reverses that challenge him.

"To him that overcometh," it is written, "will I give to eat of the hidden manna."

THE FAMILY DOCTOR AND THE GENERAL PRACTITIONER

The current discussion regarding the role and prestige of the general practitioner has resulted in a certain amount of confused thinking. Some say that he is on his way out and the sooner the better, for things are done differently nowadays than formerly. Others bewail his decline, realizing that he is the most important cog in the organization for the care of sick people.

What are the reasons for these divergent views? They are concerned with semantics, with economics and with the American obsession to organize and classify.

As to semantics, there is as much confused thinking in the medical profession as in the laity regarding the meaning of the term general practitioner. For purposes of discussion let us assume that a general practitioner is the first-echelon unit of medical care to whom the patient turns when he falls ill — whether such illness is a fever, vague fatigue and boredom with life, a perforated peptic ulcer or a fractured femur. If the patient lives in a small town situated far from a large city, the general practitioner will care for all the above illnesses. He has to be an astute internist to diagnose the fever, a skilled diagnostician and wise psychiatrist to deal properly with the tired patient, a competent surgeon to diagnose correctly and to operate upon the patient with an acute abdominal emergency, and an able orthopedic surgeon. But if the patient lives in a large city, who can deny that the best care requires that the general practitioner be primarily a medical man, and that surgical technics are of a higher order when practiced by one doing nothing else but surgery? Here the general practitioner, though still the first-echelon unit of medical care, becomes a family doctor who knows how to diagnose and deal with problems but who refers patients requiring technical procedures and special skills to those who practice such procedures every day. In the small isolated community the family doctor and general practitioner are by geographical necessity one and the same. In the large city the general practitioner required by the small isolated community should not exist — he becomes the family doctor or family medical advisor.

Economics enters into the problem in that human beings put a high value on something that can be seen or felt — something producing quick action. They will gladly pay a large sum to a surgeon to take out an appendix or hitch up a uterus, in a mistaken attempt to cure a gastrointestinal neurosis. But they will feel imposed upon if asked to pay ten or fifteen dollars to a physician who spends an hour discussing personal problems and trying to teach the patient how to deal with them himself. The fact is that compensation for medical services is based on what the public values, not

actual scientific valuation. A high premium goes with surgery, with anesthesia, with injections, with anything that the patient can grasp as presenting some definite procedure. A low premium goes with diagnostic skill, commonsense advice or friendly support. Even medical men, being themselves human, are drawn into the same way of thinking.

In discussing the problem at a meeting a surgeon recently said,"You mean the general practitioner is just a medical man who refers everyone to some specialist who can treat him?" In actual fact the family doctor can and should handle 80 to 90 per cent of the patients who consult him. But to treat them adequately he must be well trained in internal medicine, he must be a very good psychiatrist, and above all he must be as interested in the patient as a person as he is in the disease that the patient possesses. To be all of these, surely, is a full-time job, one that does not permit the time to acquire technical skills involved in surgical maneuvers. If economics is the prime obstacle to the attraction of men into this branch of medicine, either the family doctor must be content with a lower financial return than his surgical brother, or a utopia must be reached where all men receive their just deserts.

Recognizing the dangers inherent in the distribution of medical care in the complicated structure of modern medical science the attempt is now being made to organize and classify everyone. The specialty boards have become so important and the possession of a certificate so highly prized that the majority of the medical students in the country are now planning their educational program to become certified specialists. But if only 10 to 20 per cent of the patients need a specialist, such a situation is ridiculous. Obviously, something must be done to point up the importance of the family doctor or general practitioner who should take full care of 80 to 90 per cent of the patients.

The establishment of the Section on General Practice in the American Medical Association and the proposal to organize a board of general practice may be steps in the right direction, but one wonders whether these are not moves based on self defense — a kind of guild or labor-union attitude — rather than a desire to advance medical science. Would it not be more appropriate to reduce the opportunities for specialization to their proper proportions and at the same time to define clearly where the function of the general practitioner or family doctor lies? In addition, should not greater steps be taken to emphasize in hospital training qualities that the family doctor most needs — sound internal medicine and sound understanding of psychosomatic problems? Should not greater attempts be made to educate the public? If these things can be accomplished, general practice or family doctoring will assume its rightful place of esteem, and he who practices this skill will lose his all too frequent inferiority complex.

DIABETES WEEK

The week of December 6 has been designated as Diabetes Week, an arbitrary unit of time in which every effort will be made to bring under treatment the estimated million undiscovered diabetic patients in the country. This is not, obviously, a task that can be accomplished in a week; the designation is for the purpose of hammering home the existence of the problem and the necessity of meeting it. Considerable assurance was required, as the councilors of the American Diabetic Association know, to launch another special week for observance. In this case at least the cause was justified, and the need may be considered as imperative.

"The Clinical Characteristics of Early Diabetes Mellitus," by Beaser, printed elsewhere in this issue of the *Journal*, is most timely in view of the task that the American Diabetic Association has set. Beaser's statistics also, taken from the Diabetes Clinic of the Beth Israel Hospital, indicate the prevalence of early diabetes and the undesirability of having it go undetected. The task is to make the public aware of the necessity for the early discovery of the disease before overt symptoms have manifested themselves.

Patients can be classified in three groups according to the nature of the first signs or symptoms by which the disease makes its presence known. In the first group are those with frank diabetic symptoms such as polydipsia, polyuria, weight loss, weakness and pruritus. This group comprises

some 34 per cent of the total. The second group, making up 19 per cent, is composed of those who first become aware of diabetes because of the onset of one or more of its complications, such as neuritis, infection and cataract. The third, or "casual" group, comprising 47 per cent of the total, is made up of those who, with diabetes unsuspected, are found to be glycosuric on an examination conducted for a purpose unrelated to any suspicion of diabetes. It is to make the public aware of the presence of this group and the means and importance of discovering the persons who compose it that Diabetes Week has been designated.

Certain facts further emphasize the desirability of early diagnosis in diabetes. The longer the condition goes undiscovered, the more severe is its course likely to be, and it is known that sugar may be present in the urine for several years before symptoms pertaining to the disease become evident. This fact also explains its apparent onset with complications in 19 per cent of Beaser's cases — the patients had had diabetes for some time but did not know it.

Single negative urinalyses, moreover, are unreliable for the exclusion of diabetes mellitus. Stated differently, in the words of the author, "Even a single slightly positive test obtained under routine conditions, even though overshadowed by one or more previous, simultaneous or succeeding negative tests, should be taken to mean diabetes unless a glucose tolerance test proves otherwise."

INCREASE IN TUBERCULOSIS

The New York Tuberculosis and Health Association has reported a sharp increase in the number of new cases of tuberculosis in that city. For the first nine months of 1948 the total increase has amounted to 12 per cent, with a 17 per cent increase in the borough of Manhattan. The 6433 new cases registered represent an increase of 660 over the same period in 1947.

This increase is viewed with some alarm, and a corresponding increase in the number of deaths is expected, 70 per cent of all new cases registered being usually in an advanced stage of the disease. The patients now hospitalized within New York City number 5485, or 11 per cent more than a year ago. Tuberculosis sanatoriums, far from continuing with the anticipated lightening of their load, are experiencing increased pressure and overcrowding.

New York's death rate for the corresponding period of 1948 was 40.4 per 100,000 population as compared with a rate of 38.9 for 1947. This represents an increase of 4 per cent.

Boston, similarly, according to an unpublished report from the Boston Health Department, has had reported 604 new cases for the first nine months of 1947 and 694 for 1948, an increase of approximately 15 per cent. The death rate in Boston, however, is close to 65 per 100,000, although some improvement in the rate is expected for 1948. The death rate for the country as a whole is well under 40.

MASSACHUSETTS MEDICAL SOCIETY

MALPRACTICE INSURANCE POLICIES

At the present time the office of the Society is receiving many requests for information concerning malpractice insurance policies and for information regarding which company issuing such policies is approved by the Society.

For the protection of the members of the Society, I believe it is best to quote from the recommendations of the Committee to Survey Malpractice Insurance as published in the *New England Journal of Medicine*, issue of December 4, 1947, under "Proceedings of the Council," as follows: "That no one company be recommended exclusively at this time."

This recommendation was adopted by vote of the Council.

H. Quimby Gallupe, M.D., *Secretary*

ATTENDANCE AT COUNCIL AND COMMITTEE MEETINGS

All too frequently those in the offices of the Society who are responsible for the execution of its official actions are impressed with the lack of information on important issues, and even opposition, to acts of the Society, on the part of fellows who have failed to acquaint themselves with the matters involved.

A more careful reading of the issues of the *New England Journal of Medicine* that pertain to Society activities, and better attendance at meetings of the Council and committees would go a long way toward clearing up these difficulties.

In order that the business of the Society may receive due attention, the Secretary will notify all district secretaries of the attendance at meetings of the Council and of those committees whose members are elected by the districts.

H. Quimby Gallupe, M.D., *Secretary*

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

NEW TYPHOID-VACCINE DOSAGE

The time-honored dosage of typhoid vaccine of three doses of 0.5 cc., 1.0 cc. and 1.0 cc. given at intervals of seven to ten days is now to be abandoned. For many years it has been suspected that larger amounts of some biologic preparations were being given than were really required. Everyone has been reluctant, however, to reduce doses without good scientific basis, for fear that a decrease in protection would result.

The United States Army Medical Corps has pioneered in the studies regarding typhoid vaccine. Just prior to the war important improvements were made in the production and testing of the vaccine, based on a large volume of research carried out at the Army Medical Center.

Using this vaccine of uniformly high quality, the Army has determined that a smaller dosage schedule is possible. Hereafter the routine will be three 0.5-cc. doses administered at intervals of seven to twenty-eight days. The Biologics Control Laboratory of the National Institute of Health has now approved this dosage, and has authorized manufacturers to make this change in the directions accompanying the packages. The Massachusetts Department of Public Health has recommended this dosage as an optional alternative since February, 1948. In line with the policy of the National Institute of Health, it is now recommended that the new dosage schedule be universally adopted. This change may be made with assurance that the immunity produced will be as good as is required. Repeated studies at the Division of Biologic Laboratories have shown that the vaccine made there is equal or superior in potency to all batches obtained to date from other laboratories for comparison.

The recommendations for revaccination have not been changed. Either a subcutaneous dose of 0.5 cc. or an intracutaneous dose of 0.1 cc., given annually, will maintain a satisfactory level of immunity.

MISCELLANY

YALE CHILD STUDY CENTER

The appointment of Dr. Milton J. E. Senn as director of the new Yale Child Study Center has been announced by President Charles Seymour. The new center, embracing all the specialties and social sciences that bear on pediatrics, will include also the famous Yale Clinic of Child Development, established by Dr. Arnold Gesell, who retired as professor of child hygiene on July 1.

Dr. Senn, a native of Wisconsin, is retiring from the position of director of the Institute of Child Development and professor of pediatrics in psychiatry at Cornell University Medical College, to assume his new post, which will carry with it the Sterling Professorship of Pediatrics and Psychiatry.

ANNUAL MEETING OF THE SILVER HILL FOUNDATION MEDICAL COUNCIL

The medical council of the Silver Hill Foundation held its annual meeting in New Canaan, Connecticut, October 15 to 17. The members, chosen to serve in an advisory capacity for a period of two years, met first in a scientific session led by Dr. William B. Terhune, medical director of the Foundation. After this meeting, the members and their wives and guests were entertained at the Country Club of New Canaan.

The Silver Hill Foundation is a psychotherapeutic unit devoted to the study, care and treatment of the psychoneuroses where patients are given brief, intensive psychotherapeutic and physiologic aid to re-establish their lives and their ability to work. The medical staff is composed of internists who, although they are diplomates of the American Board of Psychiatry and Neurology, are interested in the eclectic approach to the treatment of the psychoneuroses rather than adhering to any one formal school of psychiatry. The subject for discussion of the Council this year was "What Is the Ideal Method of Treating the Psychoneuroses, Psychosomatic Illnesses and Psychosocial Disorders?" Speakers were Dr. Harold L. Amoss, Dr. Arlie V. Bock, Dr. Walter O. Klingman, Dr. F. C. Redlich and Dr. Edwin G. Zabriskie.

ADVISORY COMMITTEE TO CHILDREN'S BUREAU

The United States Children's Bureau, now a unit in the Social Security Administration of the Federal Security Agency, has organized an advisory committee to aid in the development of its programs for maternal and child health and crippled children's services. The Bureau is responsible for administering the $18,500,000 in grants that Congress makes available each year to the states to extend and improve these services.

The committee of forty-seven advisers, appointed by the chief of the Bureau for a three-year term, includes the following New England members: Harriett M. Bartlett, associate professor of social economy, School of Social Work, Simmons College; Dr. Allan M. Butler, professor of pediatrics at the Harvard Medical School; Dr. William T. Green, head of the Department of Orthopedic Surgery of the Children's Hospital, Boston, Massachusetts, representing the American Academy of Orthopedic Surgery; Dr. James R. Miller, of Hartford, Connecticut, trustee of the American Medical Association, representing the Association; and Dr. Duncan E. Reid, professor of obstetrics at the Harvard Medical School.

CORRESPONDENCE

DIABETES DETECTION DRIVE

To the Editor: Will you kindly publish the following open letter to the Physicians of America:

The finding of the million cases of unknown diabetes in this country poses a direct challenge to the American doctor. It is within his power to accomplish this feat. The existence of these cases in the United States has been demonstrated through a series of surveys, the most recent one conducted by the United States Public Health Service. The results of these studies now provide a springboard for organized medicine and a golden opportunity for physicians to seize the initiative *on their own* in this significant phase of public health.

The American Diabetes Association has planned a campaign to promote the early discovery and prompt treatment of the million undiscovered cases of diabetes. This campaign is unique in professional service, for according to plan the physician himself will be at the helm. Therefore, the plan cannot be prosecuted, or even started, without the endorsement and support of the entire medical profession through its governing bodies, national, state, county and local medical societies.

The plan proposed by the Association is simple, direct and sure. Through local diabetes associations, related to the American Diabetes Association and with the cooperation of local, county and state medical societies over the United States and Canada, it is planned to carry out blood-sugar screening tests by a new five-minute micro-blood-sugar method with simultaneous urinalysis for sugar with attention to the time in relation to the preceding meal. The procedure can be carried out apart from a formal laboratory. The only provision will be that the candidate must name a physician or clinic to which the results of the tests will be mailed for interpretation

to the patient. *Under no condition* will a report be sent directly to the examinee. The effort is to bring the patient with unknown diabetes under his own physician's care. There will be no statistics and no red tape.

Simultaneously, the American Diabetes Association will carry on an intensive educational campaign directed first toward doctors' postgraduate courses. It will be directed toward the layman by radio, newspapers and other publicity channels in addition to the *A. D. A. Forecast*, the Association's bi-monthly magazine, which brings to the diabetic patient homespun articles on the disease by eminent authorities in the field. At the same time the Association will place in the hands of physicians over the country an authentic *Handbook of Therapy*. Containing the most up-to-date information available, the *Handbook* will assist the physician in treating diabetic patients.

The week of December 6-12, immediately following the interim meeting of the American Medical Association, will be proclaimed as "Diabetes Week." This will be the formal beginning, the kick-off, of the Association's Diabetes Detection Drive. From this start, the program will continue on a long-term basis.

The Association is determined to do its part in finding these million diabetic patients and guiding them to you, their physicians, for treatment. May we count on your support when the matter comes up before your county or local medical society? The success or failure of the Diabetes Detection Drive depends upon you. *You* stand at the helm; this is *your* project.

HOWARD F. ROOT, M.D., *Chairman*
American Diabetes Association, Inc.

MEDICINE IN POLAND (1948)

To the Editor: Assigned to the American Embassy in Warsaw, Poland, on January 2, 1948, as attending surgeon, I had the opportunity of observing and working with Polish doctors for six months. It was necessary for the welfare of the people under my care that I become familiar with the intricacies of the hospitals in Warsaw, that I meet physicians from the different specialties, and that with our own facilities and theirs appreciated a scheme of medical treatment be worked out. My job was a pleasure, and I have many fond memories of time spent with Polish colleagues. It was an academic revelation to visualize their medical setup.

Formal comparison of American and Polish medicine is without real significance. It is of value that the physicians of an international United States of America know the level of medical education, medical care and the type of scientific endeavor in Poland. Understanding can help our outgoing aesculapiads and organizations better to deliver supplies, lecture tours and fellowships, as well as to evaluate better any Polish physician who might manage to leave Poland to apply for licensure in the United States of America. A recent bill before Congress to extend the G. I. Bill of Rights to allow servicemen to enroll in European medical schools makes it seem urgent that a spotlight be thrown on what really exists in these traditionally famous places of learning.

Poland is in general badly smashed after six years of struggle. It was smashed in such a way that its cultural and professional activities were made to suffer most. Eighty to 90 per cent of Warsaw was reduced to rubble — its hospitals, libraries and physicians barely survived the attack. In the Warsaw uprising of 1944 the medical facilities were smothered as 250,000 Poles died. Cracow, always the Polish Vienna, was luckier. Untouched physically, the manpower pipeline and scientific nourishment were cut off. Lodz, Poznan, Wraclaw, Gydynia and Lwow were all punished to some degree — perhaps Warsaw represents a rather high average picture of where medicine stands in Poland today.

In the year following the Allied victory UNRRA gave Warsaw a running start with equipment basic and for daily expenditure. Shortly thereafter the World Health Organization set up its offices and meted out fellowships to Poles eager to sail west across the Atlantic. Many were given the opportunity to go to England, Sweden and Switzerland in like manner; and WHO, together with the Unitarian Committee, sponsored visits from world-renowned specialists, who worked hard on arrival to cover the missing years of scientific improvement. Even now, 15 physicians from England, Sweden, Czechoslovakia and America are touring Poland representing the Unitarian Committee, and their success so far has been extremely gratifying. Dozens of voluntary philanthropic organizations, such as Brethren Service, Catholic Welfare and the Quaker Mission, continue to advance supplies to Poland's barren armamentarium. The seemingly insurmountable barriers to accomplishment of these missions — finance, customs and politics — were and are being overcome slowly. In like manner is the westward approach to science displacing the old Vienna school of thought and practice.

Against this background one can look out on the field of medicine in Poland, but like the sailor in *Gulliver's Travels*, one must consider whether he is amongst the Lilliputians or Brobdingnagians. It takes time to adjust the sights. It is not merely a question of evaluating the various specialties of medicine. An appraisal of their integration, so vital in American science, must be carried out in detail to arrive at an understanding of where the healing art stands in this land, the battleground between the West and Eurasia.

Unlike Germany, at one time medically self-sufficient, Poland, perhaps since the days of Copernicus, Von Mikulicz and Rydiger, has relied on the importation of science. Thus, most of the men I learned to know and work with spoke either English or German, and often many other foreign tongues. As importers and followers they seldom seem to have excelled. Where greatness existed in a few, schools apparently failed to develop or to foster progress. Under constant duress Polish medicine, along with Poland, failed to gain speed. Facilities remained limited, and ambition to renovate was stifled by traditional professorialism. The backbone of Polish medicine, hardly sturdy before the war, was scourged by an invader nation, which overtly attempted to reduce the rank of this profession, among others of the noblest elements of Poland.

I was carried into the realm of Polish surgery at the Child Jesus Hospital (First Surgical Clinic of Warsaw University) under the direction of Professor Tadeucz Butkiewicz. I was able to work two or three mornings a week in the operating room and to engage in the ward rounds and in some of the conferences. I saw 100 general surgical beds handled by eight or more assistants (men or women averaging thirty or more years of age), most of whom had at least one other job that consumed afternoons and evenings and made their work at the clinic, for a nominal sum, merely a small part of their day. Students were taught on the run and entered into the scheme of things only superficially. Teaching and academic pursuits were borne, when time permitted, by one or two. My friend Dr. Jan Nielubowicz slept little in his devotion to his role of major domo in this realm.

From this department I became acquainted with the roentgenologists and some of the internists and surgical specialists, such as Professor Gruca, a very well trained orthopedist. I found these other fields of medicine more properly tilled, although ancient devices continued to slip into recognized modern forms of therapy. Pneumonias were treated with penicillin and thoracic suction caps called "bankis," because "das alte recht" still clung like a scab to the back of men's minds. But over this lay the pall of no interdepartmental co-operation. I saw that each professor held sway in his realm, irreproachably right, seeking consultation only when a severely ill patient demanded assurance.

In every department of the hospital supplies were short. Much of what they had had been donated by UNRRA and other charitable organizations. Swedish, German and American equipment rounded out the few materials manufactured in Poland. It seemed that politics dictated amounts received from central-government purchases. Yet the wards were fairly tidy and clean, and nursing attendance was quite adequate. No great technical skill was needed in the handling of ward work because many modern devices were as yet lacking.

Results in surgery in this clinic were fair, but much was left undone owing to lack of technic, equipment and knowledge in preoperative and postoperative handling. Follow-up observation was inadequate, so that long-range errors unrevealed did not add to the standard of work. The anesthesia relied on was not only appalling, but in itself limiting. When endotracheal anesthesia was introduced by Dr. Olle Friberg, of Stockholm, its application appeared like a rainbow after a dreary rain. There was every indication that, given opportunity, the Poles could come to the fore. There was something to be noted in their traditional professional integrity, which allowed a man to carry on with pride though hindered

from every angle and which allowed even the most trying operation to be performed with practical bravado.

With 500 to 600 beds available in the Child Jesus Hospital the services seldom seemed taxed, although on several occasions there was some overflow into the halls. It was never clear how much of Poland was covered by the 15 or so hospitals in Warsaw, but certainly in some special fields patients converged on clinics here from places 300 to 400 kilometers away, even from cities where medicine was in itself quite capable.

Infrequently, I had the opportunity of visiting other hospitals. At the Wolski Hospital, Professor Landau clearly demonstrated on his sparkling medical service that he had been practicing "western medicine" since 1928. Dr. Manteuffel in surgery had operating rooms under his surveillance that lacked nothing in equipment, and his reputation in thoracic surgery seemed well deserved.

The few private clinics in which I attended patients now and then did not smack of good medicine, with nursing service makeshift and surgery performed in ancient rooms with such assistance as the attending man might be able to summon if an emergency case appeared. Anesthesia was heartrending in any event. These, then, were the sites where underpaid professors and privileged assistants rounded out their salaries, not happy in their lot, but unable to lift up the scale, being on the pan constantly themselves.

Thus, it was apparent that, as in any other plan of life, varying levels were attained, and medicine and surgery at times even though hard pressed could in capable hands rise above drab surroundings.

When I was permitted to attend the second annual (second since the war) surgical congress in Cracow, April 5-8, 1948, the surgical perspective of this struggling land was revealed. Here 300 of Poland's surgeons gathered, avidly attending Dr. Nowicki's and Dr. Glacza's early morning operating stints, after which they crammed into a small lecture hall in the well preserved Cracow city hospital for the presentation of papers. Although the orthopedic specialists were to meet separately in the following days, they attended, making the conglomeration complete, and as one of my friends complained as we strove to view the operative site early one morning, "Very few men who could wield a knife were absent."

And yet they seemed to have accomplished little after the heat of enthusiasm had waned. One and a half days were spent in uncritical bickering about treatment of wounds, centering on the role of the sulfonamides and other chemotherapeutic agents. The remainder of the second day lingered on while the surgical treatment of tuberculosis, with endless discussions on the types of operations giving best results in putting the lung at rest, was proffered. Then the mad scramble of the last day, when a rush of unrelated, crowded-out topics appeared, with the scheduled aspirants hoping to find time for their literary work. A few interesting cases were presented, but by and large the series were small with little follow-up observation. Issues were raised that had been better discussed with evidence measured in other lands, raised in some instances on spur-of-the-moment investigation. None of the papers bore the earmarks of intensive investigative work. There were no papers on medical-education or surgical-training plans. I was consoled when it became obvious that my friends were no less weary of the haranguing and no less disappointed, although "it was better than the last meeting." They were anxious to learn how a similar surgical congress was handled in America.

I had the opportunity of meeting Duzend Chorobski at this congress, and on my return to Warsaw began to visit his neurosurgical clinic in the Praga portion of town east of the Vistula river. Vigorous and esteemed, having studied and worked in many centers in Europe and in Montreal and Chicago, he demonstrated how much a well trained man could do even in difficult surroundings. Besides turning out much good surgery, Jerzy Chorobski had created a school of neurosurgery amidst ruins and already had two disciples abroad, one in Lodz and another in Cracow. He was constantly buoyed up by the generous gifts of American neurosurgeons — buoyed up spiritually as well as supplied with the physical goods needed to run his service.

In these various clinics mentioned medical students worked, some lucky enough to be allowed to do practical things, in most instances spending much of their time looking in on rounds; in surgery they never approached a sterile field. They complained bitterly, and their few friendly teachers bemoaned the large numbers in general and the expanding female representatives. Their preclinic years were ordinary — once out in the clinic they matured under the existing plan of advancement only after six to eight years. After graduating most of the class turned toward the smaller provincial hospitals for training, where they became eligible after two years to write a thesis for their Polish M.D. A few tough enough to endure obscurity for some years in Warsaw's larger clinics entered into these more academic pursuits.

Lack of reading facilities and medical literature rounded out the difficulties of education; even a master of library technic was often balked in his attempt to hunt up poorly catalogued literature, much missing in confusion and in fact. All libraries closed at eight in the evening, with little help to keep them running.

And where, after all these things were measured, did the Polish patient stand? Theoretically, Poland has a highly developed system of health centers with special setups for tuberculosis control and venereal-disease treatment — much of this exists on paper only. The average worker to enjoy medical care pays 15 to 25 per cent of his earned wage. The peasants, theoretically covered by insurance, were pretty much on their own, and many had to save up for months to pay for treatment of a medical ailment that in the meantime ran its course. They spent many earning hours waiting for care in understaffed clinics. The underpaid physicians, obliged to care for those registered, tended to give better attention to those well off, not covered by an insurance plan, who were willing to pay a reasonable fee. Even in a hospital patients were faced with the frequent necessity of obtaining their own medication on the "open market." Home-cooked food littered the wards to supplement the hospital's meager diets. Dentists, well thought of by most Americans seeking their aid, were overworked and charged a great deal, so that teeth examined in the workers were in the majority either naturally good or from neglect carious. The Poles attending our American hospital in Warsaw seemed amazed at our cleanliness, our armamentarium of drugs and our new equipment.

While I was in Poland, I saw the Unitarian Hospital in Piekary, near industrial Katowice, grow up. I heard of plans to start a large medical center in Lodz. I saw medical students being turned out to ease the burden being borne by 6000 doctors caring for 24,000,000 Poles. I heard of continued efforts to create a system of health centers centrally supported. But above all I had a chance to talk to many young doctors who saw the failure of their old professorial system and saw the need for fundamental research, as well as for better medical education and libraries, and these men realized that the West points the way to better medicine. At times they saw that old German dictums and much of Germany's arbitrary medical contributions and outmoded methods had not only led to the downfall of German medicine but also had lagged in progress while a new system of science with perhaps some humanism had grown up to the north and west.

It was all too clear that although daily changes seem to affect the practicing doctors little, their involvement in the catastrophic decline of the nation was complete — be it aggressor or aggrieved. Although Poland demanded consolation by its very plight, it demonstrated how there can prevail a communion of aesculapiads that should be fostered and nourished and looked to for the establishment of world health and peace.

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BOOK REVIEW

Telepathy and Medical Psychology. By Jan Ehrenwald, M.D. With a foreword by Gardner Murphy. 8°, cloth, 212 pp. New York: W. W. Norton and Company, Incorporated, 1948. $3.00.

This book is composed essentially of two parts: presentation of proof that telepathy exists as a phenomenon distinct from other psychic processes; and a presentation of the thesis that telepathy is in part or whole the basis of psychopathology.

To prove the first assumption, statistical data and material from case studies are presented in an attempt to put the study

on a scientific basis. In considering statistics, one must remember that they do not by themselves prove a thesis. For a thesis to become a science, two factors must be present: one must be able to predict behavior or performance from the acquired data, and the laws presented must apply to all cases that fall within the limits set up by the thesis. In no way does telepathy fill these requirements since one cannot predict its performance, and there is no explanation of the mechanism of how it works.

Psychiatry in general has fallen into the error of attempting to present itself as more nearly a science by making comparisons with well established scientific, usually physical or chemical, laws. This book at times does the same thing. One must remember, under these circumstances, that comparisons are only assertions of belief and that comparison in, and of itself, proves nothing and does not make so subjective a field as psychiatry any more scientific than if it were not made.

The second thesis is not documented with a sufficient number of cases. Certainly, in a phenomenon so subjective as the products of mental and emotional functions of the central nervous system, wherein even hallucinations appear to be real, the occasional occurrence of the same thought at the same time to two or more people can be proof of little more than chance. The fact that patients produce material similar to the thoughts of the analyst is not to be wondered at, and has been discussed in numerous books and papers and need not be detailed here. One can only use it to support a belief in telepathy if one believes in telepathy as the dynamic principle. To say that distortion of personality leading to psychoses and neuroses or other functional disturbances is based on telepathic transference of thoughts and feelings is but begging the point. One still has not explained why one person reacts to such influences, if there is such a reaction, and another does not.

The explanation advanced is far too simple. The continuing stimuli from one's environment and from within one's self are so numerous and so varied that personality is always the complex of millions of interlacing threads. One can scarcely ever hope to find the basis of personality disorders in one general underlying principle.

Finally, after the book has been read, an attempt at objective evaluation suggests that little has been added in the way of proof of existence. One still must believe or not according to one's personal bias. As each new section is begun the reader hopes that he is going to have meat to chew on, but at the end of the section there is little more substance than a mouthful of spun candy bought at a fair.

NOTICES

ANNOUNCEMENT

Dr. Morris Courtiss announces the removal of his office to 314 Commonwealth Avenue, Boston.

Dr. Manuel Kaufman announces the removal of his office from 419 Commonwealth Avenue to 1093 Beacon Street, Brookline, for the practice of internal medicine.

NORFOLK DISTRICT MEDICAL SOCIETY

A meeting of the Norfolk District Medical Society will be held at the Hotel Kenmore, Boston, on November 23, at 8 p.m.

The program will consist of a "Legislative Meeting," at which members will meet incumbent and recently elected members of the State Legislature.

Physicians and members of the Women's Auxiliary of the Norfolk District Medical Society are invited.

NEW ENGLAND DERMATOLOGICAL SOCIETY

A regular meeting of the New England Dermatological Society will be held at the Boston Ciy Hospital, Skin Out-Patient Department, at 2:00 p.m. on December 1.

These meetings are open to members and invited guests only.

INTERNATIONAL CONGRESS ON RHEUMATIC DISEASES

The seventh International Congress on Rheumatic Diseases and the first to be held in the United States will take place at the Waldorf Astoria in New York City May 30 to June 3, 1949, inclusive. This congress is sponsored by the International League against Rheumatism. The host is the American Rheumatism Association in co-operation with the New York Rheumatism Association.

Seven scientific sessions are planned, and five one-hour round-table conferences on various clinical topics will be held under the leadership of authorities in the respective fields. Short clinics, papers and reports will be given concurrently at four or five New York hospitals during three afternoons. Evening entertainment will be provided. The registration fee is $10.00.

Members of the International, the European, and the Pan American Leagues against Rheumatism as well as the Canadian Rheumatism Association, British Empire Rheumatism Council, the Heberden Society of London, and the ten state or city Rheumatism Societies affiliated with the American Rheumatism Association are especially invited.

SOCIETY MEETINGS AND CONFERENCES

Calendar of Boston District for the Week Beginning Thursday, November 25

Friday, November 26

*9:00 a.m.–12:00 m. Combined Medical and Surgical Staff Rounds. Peter Bent Brigham Hospital.

*12:00 m. Clinical Conference on Urology. Margaret Jewett Hall, Mt. Auburn Hospital, Cambridge.

Monday, November 29

*12:15–1:15 p.m. Clinicopathological Conference. Main Amphitheater, Peter Bent Brigham Hospital.

5:15 p.m. Staff Meeting. Harris Hall, New England Deaconess Hospital.

Tuesday, November 30

*12:15–1:15 p.m. Clinicoroentgenological Conference. Peter Bent Brigham Hospital.

*1:30–2:30 p.m. Pediatric Rounds. Burnham Memorial Hospital for Children. Massachusetts General Hospital.

Wednesday, December 1

*11:00 a.m.–12:00 m. Medical Rounds. Amphitheater, Children's Hospital.

*12:00 m.–1:00 p.m. Clinicopathological Conference (Children's Hospital). Amphitheater, Peter Bent Brigham Hospital.

2:00 p.m. New England Dermatological Society. Skin Out-Patient Department, Boston City Hospital.

*2:00–3:00 p.m. Combined Clinic by the Medical, Surgical and Orthopedic Services. Amphitheater, Children's Hospital.

*Open to the medical profession.

October 1–May 20. Metropolitan State Hospital. Page 418, issue of September 9.

November 23 and 30. Boston City Hospital House Officers' Association. Page 762, issue of November 11.

November 17–January 26. Boston State Hospital Psychiatric Seminar Schedule. Page 762, issue of November 11.

November 20–23. American Academy of Pediatrics. Annual Meeting. Chalfonte-Haddon Hall Hotel, Atlantic City, New Jersey.

November 23. New England Society of Anesthesiologists. Page 762, issue of November 11.

November 23. Norfolk District Medical Society. Notice above.

November 30. Hampden District Medical Society. Page 492, issue of September 23.

December 1. New England Dermatological Society. Notice above.

December 2. Suffolk Censors' Meeting. Page 492, issue of September 23.

December 4. American Federation for Clinical Research. Page 644, issue of October 21.

December 4–9. American Academy of Dermatology and Syphilology. Page 728, issue of November 4.

December 6–8. American Academy of Allergy. Fifth Annual Meeting. Chalfonte-Haddon Hall Hotel, Atlantic City, New Jersey.

December 7–9. Southern Surgical Association. Annual Meeting. Page 543, issue of April 8.

December 9. Recent Advances in Thoracic Surgery. Dr. Joseph P. Lynch. Pentucket Association of Physicians. 8:30 p.m. Haverhill.

December 9 and 10. New York State Society of Anesthesiologists. Page 34, issue of July 1.

February 4, 1949. American Board of Obstetrics and Gynecology, Inc. Page 244, issue of August 5.

March 7–9, 1949. American Academy of General Practice. Page 728, issue of November 4.

March 28–April 1, 1949. American College of Physicians. Page 158, issue of July 22.

May 16–19, 1949. American Urological Association. Biltmore Hotel, Los Angeles, California.

May 26–28, 1949. American Goiter Association. Hotel Loraine, Madison, Wisconsin.

May 30–June 3, 1949. International Congress on Rheumatic Diseases. Notice above.

November 11–17, 1949. Third Inter-American Congress of Radiology. Page 158, issue of July 22.

(*Notices concluded on page xvii*)